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AN APPRAISAL OF THE ELECTROCARDIOGRAPHIC EXERCISE TEST

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A good history is usually sufficient to establish the diagnosis of angina pectoris or coronary insufficiency. At times, however, the pain is atypical and the history doubtful; objective confirmation of the diagnosis then becomes desirable. In such cases, the electrocardiographic changes seen in response to exercise may provide this confirmatory evidence.

HISTORY OF THE EXERCISE TEST

In 1908, Einthoven[1] recorded the first electrocardiogram (ECG) after exercise; he noted the change in the ECG of his old laboratory porter after he had climbed a few stairs. The following year Nicolai and Simons[2] reported the changes after exercise in a patient who had angina pectoris. Changes occurring spontaneously during attacks of angina pectoris were recorded by Bousfield in 1918,[3] and Cowan and Ritchie in 1922.[4] In those early days of electrocardiography the changes after exercise were sometimes recorded fortuitously, since several flights of stairs had to be climbed to reach the electrocardiographic laboratory.

In 1931, Wood, Wolferth and Livezey[5] first specifically used the test to provoke attacks of angina pectoris; they investigated the changes both in normal subjects and in patients with angina pectoris. Although no untoward occurrences took place, they considered it a dangerous procedure 'to induce anginal attacks indiscriminately'. It was in 1932 that Goldhammer and Scherf[6] first recommended the use of moderate exercise followed by an ECG as an aid in the diagnosis of cardiac pain. Further observations were published by these authors in the following year,[7] and subsequently by many others.

In 1942, Master and his associates[8] standardized the performance of the test, which is now known as the Master 2-step exercise test.

THE PERFORMANCE OF THE EXERCISE TEST

There are 2 methods of performing the exercise test — a standardized and a non-standardized method.

The Standardized Method

Master and his associates[8] standardized the test according to sex, weight and age. Using these parameters, tables were constructed, based on the return of blood pressure and pulse rate to normal within 2 minutes. The exercise is performed on a special standardized 2-step apparatus and the patient is required to do a certain number of ascents and descents in 1½ minutes.

These principles may well be questioned, for it has not been shown that coronary-artery disease, once present, runs a course which is more severe in the female; nor has it been shown that ECG changes following exercise parallel those of pulse rate and blood pressure. Furthermore, although coronary-artery disease is usually more prevalent and more marked in the older age groups, in the individual as such it may be very severe in a woman of 40 years and not at all evident in a man of 80 years. According to Master's criteria, a man aged 50 years and weighing 160 pounds should do 20 ascents on the standardized apparatus in 1½ minutes, whereas a woman of the same age and weight is required to do only 16 ascents, despite the possibility that the coronary-artery disease may be so advanced in the man that only a few steps may bring on angina, whereas the particular woman may be capable of undergoing much greater exercise and thus require far more than 16 ascents before either ECG changes or angina become manifest. In addition, other factors, such as emotion and training, may influence the outcome of the test and will affect any attempt to standardize it.

More recently, Simonson and Keys[9] recommended a double Master 2-step exercise test — twice the number of ascents recommended, over twice the period. The diagnostic criteria remain the same. This further underlines the difficulties and questionable value of a standardized procedure.

Nevertheless, although the validity of the exercise test as described by Master may be questioned, it should be stated that the Master 2-step test is recognized in many centres and is commonly used as a routine procedure in many ECG laboratories.

The Non-standardized Method

Scherf recommended originally,[7] and still recommends today,[10] that the amount of exercise the patient is required to perform be adapted to the needs of the particular individual. The patient is subjected to approximately the exertion that has been known to bring on an attack of angina pectoris. This does not mean that the patient is exercised indiscriminately until such time as he develops pain. If, for example, the patient has pain after only the slightest exertion, he may be asked to do a few knee-bends or sit up and down a few times; whereas a patient who has pain only after severe exertion may be asked to climb several flights of stairs rapidly. The form of exertion is unimportant, since the object of the test is to increase the demand for coronary blood flow where an inadequate flow is suspected; the induced coronary insufficiency and resultant inner-layer myocardial ischaemia or injury may precipitate ECG changes that are diagnostic.

If no changes are noted, and if the patient's condition warrants it, the exercise test may be repeated after a suitable interval (usually 1 hour) with a cautious increase in the amount of exercise.

The following procedure is thus recommended in the performance of the exercise test:

1. The patient must not be in pain. The history and physical examination must not suggest an impending myo-

cardial infarction or acute pulmonary embolism. The patient must not be in congestive cardiac failure.

2. An ECG is recorded at rest and must be normal or at most equivocal in respect of coronary-artery disease. There must be *no* tachycardia.

3. The test is preferably performed before a meal, since physiological variants are more likely to occur after a meal. If the patient relates a history of angina pectoris after meals, the test should then be performed before a meal and, if negative, repeated after a meal.

4. The exercise test is performed according to the *non*-standardized method.

5. If pain, substernal discomfort, a feeling of faintness, or pallor develop during the performance of the test, the exercise is stopped immediately. Exercise to the point of pain is hazardous and unjustifiable. The attendance of a physician is mandatory.

6. The ECG is recorded immediately after the exercise and at 2-minute intervals for 6 minutes, or until such time as it returns to the resting configuration.

7. The ECG changes should be observed in at least 1 precordial and 1 extremity or bipolar lead. Changes in the precordial leads are usually best seen in those with the tallest R waves — commonly V_4 or V_5. Changes that occur in standard lead I are usually the most significant of those seen in the standard leads.

THE INTERPRETATION OF THE EXERCISE TEST

There are certain ECG changes following exercise which are always pathological, and others which can only be regarded as normal physiological variants. Nevertheless, the transition between what is normal and abnormal is extremely difficult, if not impossible, to define, and there is a considerable degree of overlap between the two. Since it is never possible to rule out false negative tests, i.e. a normal ECG does not necessarily exclude coronary-artery disease, it is best to establish stringent criteria in order to avoid labelling borderline physiological variants as abnormal.

Changes following exercise may affect all components of the record — the P wave, the PR segment, the QRS complex, the ST segment, the T wave and the U wave; in addition, abnormal rhythms may occur (Table I). The changes that occur after exercise are the same as those that may be seen during spontaneous pain.

Changes Affecting the P Wave

Following exercise there is a tendency for right axis deviation of the P wave, so that the P waves in standard leads II and III tend to become taller. These are normal physiological variants.

Changes Affecting the PR Segment

Atrial depolarization is normally followed by atrial repolarization, i.e. as a T wave follows the QRS complex, so a corresponding 'T' wave normally follows the P wave. This atrial T wave is known as the Ta or Tp wave and is normally opposite in direction to the P

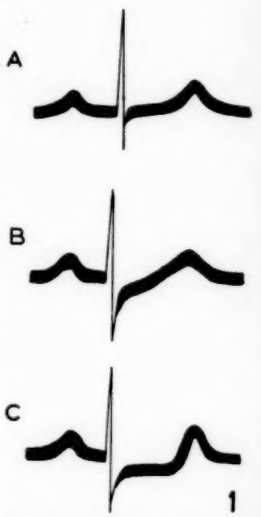

Fig. 1. Diagrammatic illustration showing: (*A*) normal QRST complex, (*B*) junctional ST-segment depression, and (*C*) plane ST-segment depression.

TABLE I. INTERPRETATION OF THE ELECTROCARDIOGRAPHIC EXERCISE TEST

Component of ECG	*Abnormal*	*Usually abnormal*	*Physiological or diagnostically uncertain*
P wave			Right axis deviation (taller in standard leads II and III)
PR segment			Downward slope
QRS complex	Left bundle-branch block	Right bundle-branch block	Right axis deviation
ST segment	Depression of 2 mm. or more in the precordial leads	Depression of 0·75 - 2 mm. in the precordial leads Any degree of plane or sagging depression	Junctional depression
	Depression of 1·5 mm. or more in the extremity leads	'Horizontality' Sharp-angled ST-T junction	
T wave		Inversion in standard lead I T in standard lead I lower than T in standard lead III Increase in height by 5 mm. or more in lead V_4 Symmetrical T waves—upright or inverted	Inversion in other leads
U wave	Inversion		
Ventricular extrasystoles	Multifocal	Unifocal: In 'showers' In bigeminal rhythm In a patient over 40 years	Isolated unifocal
	Post-extrasystolic T-wave change Post-extrasystolic U-wave change		

wave. It is usually masked by the ensuing QRST deflection and is therefore best seen following *isolated* P waves such as are found during periods of complete heart block (Fig. 3).

Following exercise, the Tp deflection normally becomes more pronounced and may cause the PR segment to have a downward slope (Fig. 4, ½-minute and 2-minute tracings). It may cause junctional depression of the ST segment, especially with short PR intervals, thus producing a false positive ST depression.

Changes Affecting the QRS Complex

There is a normal tendency for right axis deviation of the QRS complex following exercise.

The development of abnormal widening of the QRS complex is regarded as a positive test. The appearance of left bundle-branch block after exercise is always pathological. Frank right bundle-branch block is usually abnormal, but may occasionally be found in normal individuals.

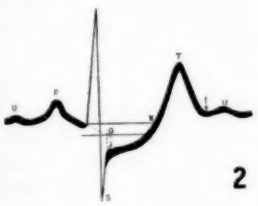

Fig. 2. Diagram illustrating methods of measuring (1) true and false depression of the ST segment, and (2) the degree of horizontality of the ST segment. (1) The line of the sloping PR segment is continued until it meets, at point O, a vertical line drawn from the junction of the QRS and ST deflections; the distance O—J indicates the true amount of ST-segment depression. (2) A horizontal line drawn from the beginning of the QRS deflection is continued till it meets the T wave at point X. This distance, Q—X, is expressed as a percentage of the QT interval (measured from the beginning of the QRS complex to the end of the T wave—arrow); Q—X is greater than 50% of QT in the majority of true-positive tests.

Changes Affecting the ST Segment

ST-segment depression is frequently seen following exercise and it is the interpretation of this sign that has caused the most difficulty and occasioned the greatest controversy.

ST-segment depression may be interpreted both quantitatively and qualitatively, i.e. both the amount and type of depression should be considered. There are 2 principal types of ST depression:

(*a*) The whole or nearly all of the ST segment is depressed, resulting in horizontality, or plane depression, of the ST segment (*C* in Fig. 1; ½- and 2-minute tracings in Fig. 4; Fig. 5). Normally, the ST segment merges smoothly and gradually with the ascending limb of the T wave, so that a definite separation between the two is difficult or impossible to define (*A* in Fig. 1; Fig. 5); the horizontality or plane depression will result in a sharp-angled ST-T junction. This form of ST-segment depression or configuration should always be regarded with suspicion and is nearly always abnormal (Figs. 1, 4 and 5). Indeed, even if there is no depression, but the ST segment has the appearance of horizontality with a sharp-angled ST-T junction, the change is usually abnormal. Measurement of the degree or amount of horizontality has been attempted by Lepeschkin and Surawicz[12] (for details, see Fig. 2).

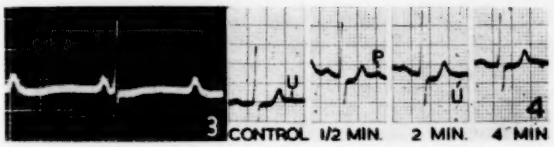

Fig. 3. Isolated P waves from a case of complete A—V block. The Tp wave can be seen as a negative deflection after each P wave. A QRS complex has been artificially placed over the second Tp deflection and illustrates how an ensuing ST segment could be depressed to the extent of 0·5 mm. by the Tp deflection.

Fig. 4. ECG recording during an exercise test; standard lead I. Note: normal upright U wave in the control tracing and an inverted U wave in the 2-minute tracing; superimposition of the P wave on the U wave during the tachycardia of the ½-minute tracing; 1·25 mm. *plane* depression of the ST segment with *sharp-angled ST-T junction* in the ½-minute and 2-minute tracings, contrasting with the smooth ST-T transition in the control tracing; the downward slope of the PR segment in the ½-minute and 2-minute tracings. There is also a prolonged PR interval in the control and 4-minute tracings. *Comment:* Correction of the ST-segment depression to allow for the false depression of the Tp deflection (*cf.* Fig. 2) reveals a true depression of 0·75 mm. This amount of depression is usually, though not invariably, diagnostic. However, the plane configuration and the presence of an inverted U wave makes this test positive and diagnostic.

(*b*) The other form of ST-segment depression affects the proximal part or junction of the QRS deflection with the ST segment (*B* in Fig. 1). This junctional form of depression should be interpreted with caution, since it may sometimes be found in the normal individual. This is from the effect of the Tp deflection (see below), which becomes more marked after exercise (*cf.* Fig. 3).

ST-segment deviation is best seen in the precordial leads with the tallest R waves — usually V_4 and V_5.

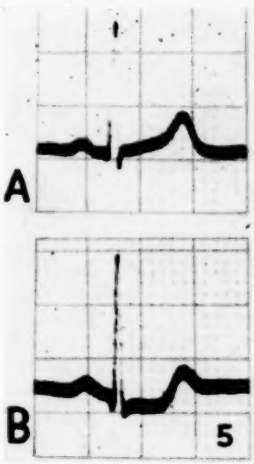

Fig. 5. A. normal QRST complex. Note the smooth transition from ST segment to T wave. *B.* QRST complex of coronary-artery disease. Note plane depression of the ST segment and the sharp-angled ST-T junction.

The amount of depression that is considered definitely abnormal is the most disputed point in the interpretation of the exercise test. Figures in excess of 0·5,[6] 0·75,[12,13] 1·0,[14-16] 1·5,[17-20] and 2[21-23] mm. depression in the precordial leads V_4 and V_5 have all been considered as definitely abnormal. The matter is further complicated by the fact that it is at times difficult to judge the position of the baseline as a reference point from which to measure the depression, and the depressing effect of the Tp deflection must also be taken into account. The best baseline or

isoelectric level to use is the UP segment (Fig. 2), but this is often obscured during the tachycardia which so frequently follows exertion (½-minute recording in Fig. 4). In such cases the baseline is measured from the junction of the PR segment with the QRS complex. Owing to the depressing effect of the Tp deflection, the PR segment may have a downward slope and the amount of ST-segment depression may be more than that caused by coronary insufficiency alone.

To avoid the inclusion of this false depression of the ST segment, Lepeschkin and Surawicz[22] have devised a procedure in which the downward slope of the PR segment is continued to meet a vertical line extended from the junction (J) of the QRS complex with the ST segment (Fig. 2). This level is taken as the true baseline and these authors, in a well-controlled investigation, have found that, by using a criterion of 0·75 mm. depression below this baseline, only 16% false positives occurred as compared with 26% using the same procedure with Master's criterion of 0·5 mm. depression. Nevertheless, 16% is still a high percentage of false positives. Other reports[23,24] have also shown that ST-segment depression of 1 mm. or more may be found in normal individuals.

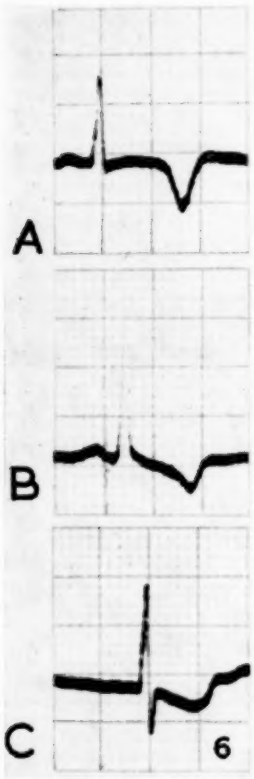

Fig. 6. A. T wave in coronary-artery disease. Note the symmetry and pointed vertex. *B.* T wave in hypertensive heart disease. Note asymmetry and absence of pointed vertex. *C.* T wave with digitalis effect. Note asymmetry and straight downward slope of the ST segment.

The position was best evaluated by Scherf[23] who stated that, since it is impossible to eliminate false negative tests, i.e. since a negative result or normal ECG does not exclude cardiac disease, it becomes imperative to avoid false positive tests, consequent incorrect diagnoses, and possible iatrogenic disease. Thus Scherf deliberately set extremely stringent criteria to avoid making incorrect diagnoses, so that a positive test should be based on incontestable standards. The test is therefore only considered diagnostically positive when the ST-segment depression is 2 mm. or more in the precordial leads and 1·5 mm. or more in the extremity leads. This is well seen in Fig. 7, which illustrates the changes occurring during a spontaneous attack of angina pectoris. Nevertheless, any depression of between 0·75 mm. and 2 mm. in the precordial leads should be regarded with suspicion, since it is usually, though not inevitably, abnormal.

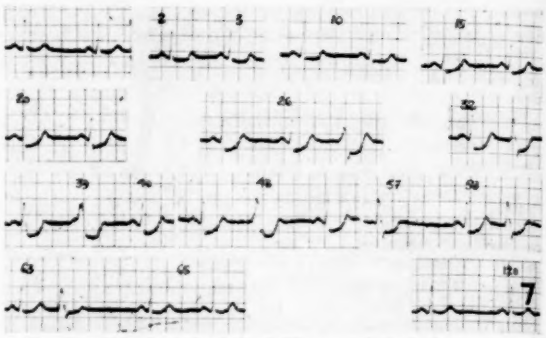

Fig. 7. ECG (standard lead II) recorded during a spontaneous attack of angina pectoris. Figures indicate the number of seconds after the onset of pain. Note the ST depression of 3 mm. during the period of 26 - 48 seconds after the onset of pain; also the frequent ventricular premature beats. This amount of ST-segment depression is always diagnostic of coronary insufficiency. It is noteworthy that the Master 2-step exercise test in this individual was consistently negative and that the changes recorded above occurred only during spontaneous attacks of pain. (Courtesy Dr. H. J. L. Marriott and The Williams and Wilkins Co., Baltimore.)

Changes Affecting the T wave

The T waves in the precordial and augmented limb leads frequently become diphasic, isoelectric or inverted, following exercise. The T wave, however, is the most unstable component of the ECG recording; changes of this deflection may occur with hyperventilation, heavy meals, anxiety, smoking, drinking iced water, changes in bodily position, and decrease in blood pressure. Variations also occur with age and race. They are found so frequently as normal variants that if they occur as isolated phenomena their diagnostic import is uncertain.

Despite this, there are certain T-wave changes that are frequently suggestive of coronary-artery disease and, although not in themselves definitely diagnostic, are pointers towards the presence of coronary insufficiency. The T wave of coronary insufficiency has symmetrical limbs and a sharp pointed vertex (Fig 6 *A*); the ST segment usually shows an upward convexity. The T-wave configuration from other causes usually shows asymmetrical limbs without a peaked vertex (Fig. 6 *B* and *C*).

In the presence of a dominantly positive QRS deflection in lead I, frank inversion of the T wave in that lead is usually abnormal.[12] Furthermore, in the presence of a dominantly positive QRS deflection in lead I, a T wave in lead I that is lower than a T wave in lead III is likewise frequently abnormal.

Occasionally the T wave becomes taller, pointed, and symmetrical following exercise. If the increase in height in lead V_4 is 5 mm. or more than the resting value, it should be regarded with suspicion and is usually abnormal.[12]

Changes Affecting the U Wave

The U wave is a small deflection occurring just after the T wave (Figs. 2 and 4). It is best seen in the precordial leads reflecting the transition zone — usually V_2 - V_4. It

is normally in the same direction as the T wave. The deflection may be so small as to make accurate recognition extremely difficult, and with tachycardia the U wave may be superimposed on the following P wave — this makes recognition impossible (½-minute tracing in Fig. 4).

An inverted U wave, i.e. a U wave which is opposite in direction to the T wave, is diagnostic of cardiac disease, especially of coronary-artery or hypertensive origin. When it develops after exercise it always constitutes a positive response and indicates cardiac ischaemia[12] (Fig. 4). Occasionally, it is the only abnormal finding.[25]

Changes in Rhythm Following Exercise

Sinus tachycardia normally follows exercise. It may occasionally precipitate left or right bundle-branch block (see above).

The presence of *multiform ventricular premature beats* is diagnostic of cardiac disease. When they develop in response to exercise they constitute a positive test.

Unifocal ventricular premature beats may occasionally be found in the normal subject. Nevertheless, their presence after exercise usually means abnormality, especially if they occur in 'showers', if they give rise to short runs of bigeminal rhythm, if they occur in a person over 40 years of age, or if they persist for several minutes or longer.

ADDITIONAL FACTORS TO BE CONSIDERED

The Duration of ECG Abnormalities after Exercise

ECG changes, particularly ST-segment depression, from coronary insufficiency tend to last longer than those caused by physiological variants. Although there are exceptions, normal variants or false-positive changes usually last less than 2 minutes, whereas pathological or true-positive changes commonly last 5 minutes or longer.[12]

A depression of, say, 0·75 mm. in the precordial leads, which is usually, though not definitely, diagnostic of cardiac ischaemia, is considerably strengthened as a criterion of positivity when the change lasts for 5 minutes or longer.

The Effect of Digitalis on the Exercise Test

The exercise test cannot be interpreted with confidence in the presence of digitalis effect. Digitalis itself may markedly influence the ST segment (Fig. 6 *C*) and positive tests have been reported in patients taking digitalis, in whom there was no evidence of coronary-artery disease.[26,27]

Hypertension and the Exercise Test

The exercise test should be interpreted with caution in the presence of hypertension, since this may at times mimic the effects of coronary insufficiency. Following exercise, a pattern of left ventricular hypertrophy — QRS changes only — may change to one of 'strain' — ST-segment depression with T-wave inversion; U-wave inversion may also occur. Such T-wave change, however, shows asymmetrical limbs without a pointed vertex (see above and Fig. 6 *B*).

Relationship of ECG Changes to the Development of Pain

While patients who develop pain on exercise usually develop abnormal ECG changes, this correlation is not invariable. The appearance of abnormal ECG changes does not necessarily correlate with that of pain. Although the patient should not be exercised to the point of pain by intent, when pain is precipitated as a result of the exercise test it may occur long after the appearance of abnormal changes and may disappear long before such changes have regressed. Abnormal changes, which cannot be reproduced by exercise, may be found during spontaneous pain (Fig. 7).

Dangers of the Exercise Test

Although the element of danger in the exercise test can never be entirely eliminated, the incidence of reported fatalities are infinitesimal and the cause-and-effect relationship often questionable. Instances of myocardial infarction precipitated by the exercise test have been

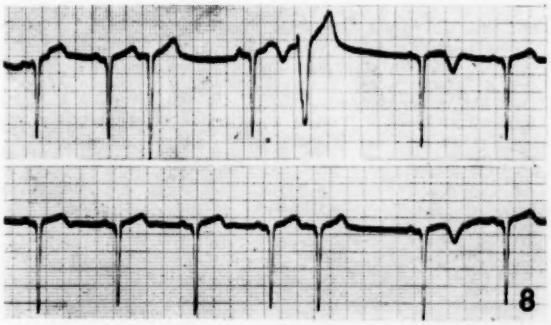

Fig. 8. ECG (lead V_1, continuous strip) showing post-extrasystolic T-wave inversion. The 3rd cycle in the upper strip and the 5th cycle in the lower strip are atrial extrasystoles; the 5th ventricular complex in the upper strip is a ventricular extrasystole. Note the marked inversion of the T wave in the normal sinus complex *following* each extrasystole.

reported where the basic requirements for the performance of the test were not fulfilled. These cases are often quoted without critical evaluation. In particular, the case reported by Grossman and Grossman[28] is worthy of comment. The patient gave a typical history of myocardial infarction 4 months previously; the so-called borderline tracing showed (*a*) a tachycardia of 100 per minute, (*b*) a sagging ST-segment depression with sharp-angled ST-T junction in standard lead II, (*c*) obvious U-wave inversion in leads I, V_2, V_4 and V_5. The diagnosis of coronary-artery disease was thus well established both clinically and on ECG, and the performance of the test was unnecessary. The case reported by Friedberg[29] likewise shows U-wave inversion in the 'normal' control tracing.

The test is reasonably safe, provided the basic precautions, outlined above, are taken.

The "Poor Man's Exercise Test"

Levine has facetiously styled the chance finding of an extrasystole with post-extrasystolic T-wave changes in the control tracing as a "poor man's exercise test", since it gives immediate evidence of abnormality and obviates the necessity for a further, more expensive, exercise test.

The abnormality consists of a T-wave change in the *first sinus beat following* an atrial or ventricular extrasystole (Fig. 8). The T wave usually becomes inverted,

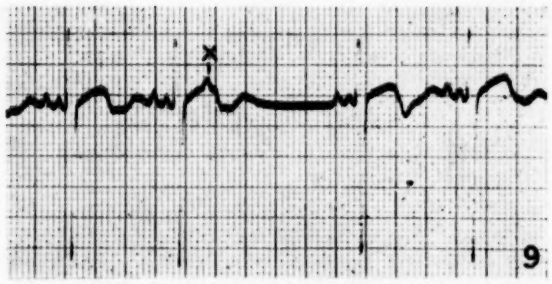

Fig. 9. ECG (lead V_1) showing a blocked atrial extrasystole — the ST segment of the second QRST complex is deformed by the superimposition of the ectopic atrial 'P' wave (X) occurring so early that the A—V nodal tissues are still refractory and therefore unable to conduct the impulse to the ventricles. Note the inversion of the T wave in the sinus beat following the blocked extrasystole. This demonstrates that it is not the extrasystole *per se* which causes the post-extrasystolic T-wave inversion, but rather the pause which it occasions. There is also intra-atrial block — P-wave duration = 0·14 seconds.

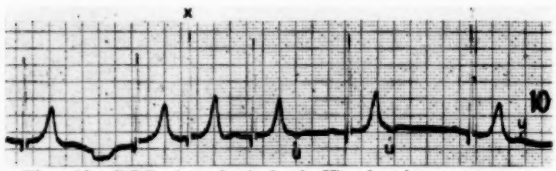

Fig. 10. ECG (standard lead II) showing post-extrasystolic U-wave inversion. X represents an atrial extrasystole. Note the U-wave inversion in the 2 sinus beats following the extrasystole.

but may become taller than normal; any change is significant. Similar changes have been observed following *blocked* atrial extrasystoles[30] (Fig. 9). Thus, it is not the extrasystole *per se* that causes this change, but rather it is the pause it occasions that evokes the change.

Occasionally, U-wave inversion may also be observed following extrasystoles (Fig. 10).

The above signs are nearly always diagnostic of cardiac disease.

I wish to express my thanks to Dr. H. J. L. Marriott and the book publishers, The Williams and Wilkins Company, Baltimore, Md., USA, for permission to use Fig. 135 from Marriott's 'Practical electrocardiography' reproduced here as Fig. 7.

I should also like to express my thanks to the Photographic Unit of the Department of Medicine, University of the Witwatersrand, for the photographic reproductions.

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SUIDELIK-AFRIKAANSE HARTVERENIGING: TAK STELLENBOSCH

Agt gevalle van bewese Coxsackie B_3 virus miokarditis by pasgeborenes is by die onlangse vergadering van hierdie tak op 24 Augustus deur dr. P. V. Suckling bespreek. Die kliniese beeld is reeds voorheen beskryf deur die spreker,[1] en verwante ondersoeke van hierdie reeks gevalle het in dieselfde blad verskyn.

Die ouderdom van die babas het van 5 tot 8 dae gewissel en almal het met koors gepresenteer. Die diagnose is afhanklik (in besonder) van 'n geskiedenis van Bornholm-siekte by die moeder, of die teenwoordigheid van 'n epidemie by die hospitaal. Koors en 'n rooi keel is die belangrikste fisiese tekens in die begin, voor hartversaking, hepatomegalie en perifere versaking ontwikkel het.

Die histologiese beeld van die miokardium en lewer is ook bespreek. Fokale nekrose is sigbaar na 7 dae. 'n Polimorfe infiltrasie is in die akute stadium teenwoordig. Na 7 dae kom fibrose voor. Virusstudies is vermeld, met beklemtoning van die kweek van Coxsackie B_3 virus wat van die hartspier by lykskouing verkry is en van die stoelgange van ander aangetaste babas. Epidemiologies is geen tekens van 'n kruisinfeksie van baba tot baba opgemerk nie.

Bespreking

In die lewendige bespreking wat op hierdie voordrag gevolg het, het prof. A. J. Brink gevra na die kliniese beeld wat as hartversaking bestempel word. Daar was tagikardie, maar die hepatomegalie is nie aan stuwing toegeskryf nie en daar was geen radiologiese of outopsie-bewys van hartvergroting nie. Die outopsie-bevindings dui op 'n miokarditis sonder versaking. Die teenwoordigheid van sentrale sianose wat opgehef is deur suurstof-toediening, het ook gespreek ten gunste van eerder 'n longaantasting, as die gevolg van 'n hartletsel. Die terapie van 'n plat ligging in hierdie pasiënte met versaking is ook bespreek, asook die waarde van 'n soutbeperking by babas waar onryp tubulêre funksie aanwesig is. Dr. Suckling wys daarop dat met ‚hartversaking' hier nie na kongestiewe hartversaking verwys word nie, maar na 'n ontoereikendheid van die kardiale spier a.g.v. die miokarditis. Die babas word plat verpleeg met die oog op serebrale anoksie en om hul stil te hou en so min moontlik te laat vermoei. Histologies kan 'n longletsel nie aangetoon word nie.

Dr. B. J. v. R. Dreyer en prof. H. W. Weber het hierna die patologiese beeld bespreek en die spreker het bevestigend geantwoord op prof. Weber se vraag na die aanwesigheid van 'n endokarditis in hierdie gevalle.

Dr. W. H. Opie het dr. Suckling bedank en daarop gewys dat hierdie epidemie baie sorgvuldig en noukeurig ondersoek is op 'n baie bekwame wyse.

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EDITORIAL : VAN DIE REDAKSIE

THE DOCTOR AND THE MEDICAL ASSOCIATION

Once again we are nearing the end of the year, and a large number of newly-qualified doctors will be joining the ranks of the medical profession. Since the first group of students will be graduating from Stellenbosch this year, all five of our medical schools are now fully productive. In view of this fact, therefore, it would be fitting to stress once more, as in the past, the importance of membership of and dedication to the Medical Association of South Africa — the only national professional organization of doctors in this country.

It has become a tradition for professional men all over the world to organize themselves into learned societies to safeguard their material interests and to provide a medium through which they can give expression to their cultural and scientific aspirations. In most of the countries of the Western world doctors have organized national medical associations which are, in turn, members of the World Medical Association. In the same way we, in this country, have established the Medical Association of South Africa 'to promote the medical and allied sciences and to maintain the honour and interests of the medical profession'.

Since the early days of its existence it has been the explicit aim of the Medical Association to function as a responsible body of professional men who are fully aware of the great and important obligation which rests on them — to keep abreast of the times in scientific and cultural matters.

Admittedly, the Association has, in recent years, been subjected to severe scrutiny and criticism. It must, however, be borne in mind that it has had to face extremely difficult problems especially in the field of the economics of medical practice. The Association can only continue to deal with these problems on a satisfactory level if it can be assured of the whole-hearted support, not only of all its members, but also of each individual practising doctor, irrespective of whether he is in private practice or in full-time employment.

The advantages of membership of the Medical Association have been well known to a large number of its members all over the country, but there are still many doctors who are unaware of these advantages. Furthermore, there are the newly-qualified doctors we have previously referred to. It is to these three groups of doctors — those who are in private practice and are not members of the Association; those who are in full-time employment, including professors, research workers, health officers, etc. who are not convinced of the value of the Association as a scientific and academic body of national importance; and those who have qualified recently — that we should like to extend a special invitation to become members of the Association. The Association is at present urgently engaged in attempting to find a formula which will attract all those who are in full-time employment to the Medical Association.

In particular, we should like to draw the attention of all doctors to the excellent article on 'The Medical Association: its rôle in the past and its ideals for the future' which was published in the issue of the *Journal* for 31 May 1960 (**34,** 423). This article was written by Dr. J. H. Struthers, Past-Chairman of the Federal Council, and deals with the services rendered by the Association to the profession in the fields of the economics of medical practice, the publication of the *Journal*, the rôle of the Association in promoting medical education in the widest sense of the word, international affiliation, and the Association's hopes for the future.

Following is a brief summary of all the services which are at present being provided by the Association:

1. Opportunities for meeting colleagues, holding scientific meetings and providing a forum for the exchange of opinions.
2. A *Journal* for the spreading of medical knowledge.
3. Means for the settlement of ethical disputes between members.
4. Means for negotiating with medical aid societies and provision of some measure of control over medical benefit societies.
5. Means for negotiating with the Workmen's Compensation Commissioner.
6. Acting as the voice of the profession in all matters concerning medical practitioners, and being recognized as the official body in various Acts and Ordinances.
7. Legal protection for individual practitioners.
8. Procuring of income-tax concessions of various kinds.
9. Obtaining preferential insurance of various forms for members.
10. Assistance to members by the Agency departments.
11. Amenities for members travelling overseas by reciprocity with the British Medical Association and the Canadian Medical Association, and through membership of the World Medical Association.
12. Improvement of salary scales of full-time personnel.
13. Influence on medical schools and medical education generally, e.g. encouraging and working towards the establishment of the College of Physicians, Surgeons and Gynaecologists of South Africa.
14. Postgraduate courses, provided directly or through medical schools.
15. Library facilities through grants to medical school libraries.
16. Assistance to needy dependants of members, through the Benevolent Fund.
17. Acting as a unifying factor, through Branches and Divisions, among practitioners.
18. Liaison with other professional bodies and the public.

The Association can succeed in playing a satisfactory and worth-while rôle in medical professional life only if it has the wholehearted support of *all the doctors* in the country.

BEELDRADIOVERTONINGS BY DIE MEDIESE KONGRES

Lede van die Mediese Vereniging wat die pasafgelope Kongres van die Vereniging in Kaapstad bygewoon het, het vir die eerste keer in die geskiedenis van die Vereniging die geleentheid gehad om in hierdie land 'n reeks beeldradiovertonings in kleur te sien. Hierdie vertonings is moontlik gemaak deur die firma Smith, Kline en French wat die beeldradio-eenheid na Suid-Afrika toe gebring het nadat die Minister van Pos- en Telegraafwese toestemming gegee het vir die vertoning van geslote-baan, mediese beeldradio-uitsendings vir gehore van geneeshere, en ander mediese personeel. Soortgelyke vertonings is ook van 9 tot 13 Oktober in Johannesburg, en sal van 23 tot 27 Oktober in Durban aangebied word.

Die vertonings bestaan uit die uitvoer en aanbieding van operasies of ander mediese prosedures deur 'n span geneeshere. Die prosedures word met beeldradiokameras afgeneem en op 'n groot doek in 'n lesingsaal geprojekteer. Die chirurg en sy assistente, insluitende die narkotiseur en ander lede van 'n spesiale paneel van geneeshere, verduidelik sistematies en stap vir stap die prosedures waarmee hulle besig is. 'n Moderator, wat in die lesingsaal is, lei die bespreking en rig vrae aan die span wat in die operasiesaal werk.

Die mediese professie het dus direkte geleentheid gehad om beeldradiovertonings as onderrigmetode te sien en te bestudeer. En ons wil dadelik sê dat die eksperiment 'n groot sukses was. As onderrigmetode vir die toekoms het dit beslis baie groot moontlikhede, veral omdat so baie soorte prosedures, bowe en behalwe chirurgiese operasies, hulle tot hierdie metode van onderrig en benadering leen.

Vir die student is daar natuurlik geen substituut vir die persoonlike, daadwerklike *doen* van dinge nie. Elkeen moet sy eie praktiese ervaring en vaardigheid uitbrei en verbeter op die grond van sy teoretiese kennis en insig. Maar, dit is ook nie die bedoeling met beeldradiovertonings om hierdie ervaring uit te skakel nie. Die waarde van die vertoning is juis om vir die voornemende beoefenaar van die vak 'n agtergrond en verwysingsbasis te skep waarop hy dan sy eie pogings kan modelleer.

Die tegniek van beeldradiovertonings skep veral die moontlikheid vir 'n ‚meester' om die fynere besonderhede van sy tegniek aan 'n groot aantal toeskouers te vertoon op 'n manier wat nie in die operasiesaal self moontlik is nie. Dit wil dus voorkom of direkte ondervinding in die operasiesaal, aangevul deur herhaalde geprojekteerde vertonings van prosedures en metodes deur die ‚meester' self, die ideale benadering tot die onderrig van baie aspekte van die medisyne in die toekoms kan vorm.

Namens die mediese professie wil ons aan die Minister, dr. A. Hertzog, die versekering gee dat hierdie nuwe metode van onderrig met 'n kritiese gesteldheid benader is. Ons is dankbaar vir die geleentheid om self direkte ervaring van beeldradio-onderrig te kon opdoen, en dit sal help om ons perspektief ten opsigte van onderrigmetodes te verbreed.

Lede van die professie wat nie die geleentheid gehad het om beeldradio-uitsendings in kleur in Kaapstad te bestudeer nie, word aangeraai om die vertonings by 'n ander geleentheid te probeer bywoon. Op dié manier sal ons dan later die menings van uiteenlopende deskundiges kan kombineer om dan te probeer om ooreenstemming te bereik oor nog 'n manier waarop ons mediese onderrig bevorder kan word.

CONGENITAL AORTIC STENOSIS

A REVIEW ILLUSTRATED BY 18 CASES TREATED BY OPEN-HEART SURGERY

DAVID ADLER and DENIS FULLER, *Thoracic Surgeons, Johannesburg*

The subject of this paper is one that until recently was thought to be rare, benign in its course, capable of differentiation between valvular and sub-valvular types, and not amenable to adequate and safe surgical correction. This presentation, on the contrary, will show that it is far from rare, can be malignant in its course, cannot readily be clinically separated into its 2 varieties, and is now satisfactorily and safely cured by direct-vision surgery with the aid of the pump oxygenator.

History

Carolus Rayger in 1672, according to Campbell,[1] first described congenital valvular stenosis in a Parisian cobbler aged 40. Chevers, according to Brock,[2] first described a case of sub-valvular stenosis in 1842. Tuffier[3] in 1913 was probably the first to attempt surgical correction of this malady in humans. In the following year Carrell[4] first suggested direct vision for surgery of the aortic valve and successfully opened the aorta for 2½ - 3 minutes in a series of experiments. Smithy,[5] himself suffering from aortic stenosis, passed a valvulotome through the aortic wall in 22 animal experiments, but haemostasis was inadequate with his methods. In the same year, 1947, Brock[6] used an operating cardioscope which he passed down the right subclavian artery. In 1952 Bailey[7] of Philadelphia first reported his technique of transventricular valvotomy.

The first direct-vision operation recorded was by Clowes[8] in 1954. He employed a pump oxygenator, but the patient did not survive. On 10 October 1955, Julian[9] successfully exposed the aortic valve under hypothermia in man. Lewis[10] and Swan,[11] in the following month, and Brock,[6] in January 1956, had their first successes under hypothermia.

It was left to Lillehei,[12] the pioneer of the bubble oxygenator, to report the first successful treatment of calcific aortic stenosis by direct-vision surgery with cardiopulmonary by-pass.

Incidence

In her classical monograph, first published in 1936, Maud Abbott[13] reported 23 cases in her 1,000 autopsies of congenital heart lesions. Dry,[14] from the Mayo Clinic, in his monograph on 'Congenital anomalies of the heart and great vessels', published in 1948, had no record of congenital aortic stenosis in the 132 cases reviewed. Paul Wood[15] in his book 'Disease of the heart and circulation' devoted half a page to this subject and stated: 'aortic valvular stenosis is rare'. McMahon,[16] in 1953, reviewed all congenital heart lesions in Birmingham over

a 10-year period, and found only 4 patients with congenital aortic stenosis among his 372 cases. On the other hand, Nadas,[17] from his extensive experience at the Children's Hospital in Boston, in 1957 reported 67 cases in 5 years. Campbell[1] reported 40 cases from Guy's Hospital. In his experience, 7% of acyanotic heart lesions and 3% of all congenital lesions are congenital aortic stenosis.

Sex Incidence

Most patients are males. Nadas[17] stated that the condition is 5 times more common in boys. Spencer[18] described 11 males out of 12, while Morrow[19] recorded 20 males out of 30. Of our 18 patients, 12 were males.

Age

Although we are describing a congenital lesion, few cases have been recognized in early infancy. However, together with patent ductus and coarctation the condition should always be considered when cardiac failure occurs in infancy. Cooley,[20] out of a surgical experience of 13 cases, operated upon 3 desperately ill, pulseless infants under the age of 6 months. Our youngest patient was aged 5 years and our oldest 46 years.

Pathogenesis

The bicuspid aortic valves with stenosis are evolved from the proximal ends of the distal bulbar swellings, and fusion could occur at this stage. Brown[21] quoted Farber and Hubbard[22] as showng cases in which foetal endocarditis was responsible for this lesion. Sub-aortic stenosis, on the other hand, according to Keith[23] and quoted by many authors, is always embryological in origin and is caused by imperfect inclusion, or persistence of part of the bulbus cordis into the outflow tract of the left ventricle. This raises an irregular ridge of thickened fibrous endocardium on the prominent muscle of the septum which crosses from there onto the ventricular surface of the aortic cusp of the mitral valve.

Pathology

Although Campbell[24] and Brown[21] stated that the valve cusps are thickened, irregular and deformed, even in children, this is not our experience, possibly because of the youth of most of our patients. Variations in size and shape of the cusps do occur. Bicuspid valves are a common anomaly and were found in 6 of our cases. A diaphragmatic dome with a central opening, such as is almost invariably found in congenital pulmonic stenosis, has been rare in our cases. Calcification occurs in the older age group, as was shown in one of our patients, aged 32, who was subjected to operation under hypothermia. In almost all our cases commissures could be detected.

Post-stenotic dilatation of the aorta distal to the obstruction is usual, and its absence suggests some degree of hypoplasia of the aortic wall. This we saw in our 12th case, where the aorta was small and its wall was almost cartilaginous in character and associated with narrowing of the valve ring. In our second case performed under hypothermia, the wall of the aorta, itself greatly dilated, was unfortunately paperlike and we lost this patient from reactionary haemorrhage from the aortotomy incision. Autopsy confirmed that she had ovarian agenesis — Turner's syndrome. The left ventricle showed marked concentric hypertrophy with a wall thickness of 3·5 cm. and a minute cavity.

In sub-aortic stenosis there is usually a fibrous diaphragmatic ring or an irregular raised ridge of thickened fibrous endocardium on the prominent muscle of the septum 1 - 3 cm. below the valves, and across from this onto the ventricular surface of the aortic cusp of the mitral valve. This fact must be reiterated — the aortic cusp of the mitral valve forms the left posterior portion of the left ventricular outflow tract and the base of the sub-valvular ring. Marquis and Logan,[25] at autopsy, have confirmed fibrosis of the left ventricular muscle fibres following death of the cells from anoxia and oedema secondary to compression by the hypertrophied muscle mass.

In our series of 18 cases, 6 patients had bicuspid valves, 4 had sub-valvular stenosis, and 4 had hypoplasia of the aortic ring.

Associated Lesions

Probably coarctation of the aorta is the most common lesion associated with congenital aortic stenosis and was present in our first case. Of Downing's[26] 37 cases, 8 had coarctation of the aorta. Pappas[27] reviewed 2 such patients operated upon by closed methods by Bailey through the left chest, and quoted 24 autopsy reports from the literature.

Patent ductus arteriosus is also quite common as an associated lesion. Bonham-Carter[28] reported 8 cases of patent ductus associated with aortic disease, but of these only his last patient had congenital aortic stenosis. We tied off a patent ductus in a child aged 6 years, and 2 years later performed open aortic valvotomy on her. In a second case a patent ductus was ligated in 1949, and at aortotomy 11 years later a hypoplastic aortic valve ring was found.

Hypoplasia of the ascending aorta is not uncommon, and a narrowed aortic ring can be one of the major problems in surgery, as we have found in 4 of our cases.

In our series of 18 cases, 1 was associated with coarctation of the aorta, 2 with patent ductus, and 1 with abnormal thoracic vertebrae.

Prognosis (Table I)

Although until recently it was felt that these lesions were benign in their outlook, this has not been substantiated by more recent critical reviews. Nadas[17] recorded 5 deaths in his 67 patients over a 5-year period. If there is left ventricular hypertrophy on the electrocardiogram the prognosis is bad in his opinion. Helen Taussig[29] stated 'most individuals live at least to adult life. The majority die of subacute bacterial endocarditis. Sudden death may occur'. In Campbell's[1] series of 40 patients 2 died suddenly while under observation. Maud Abbott[18] recorded that the average duration of life in all cases of valvular stenosis was 3½ years, whereas in 12 cases of sub-aortic stenosis it was 22½ years. Brock[3] stated that there is no difference in prognosis between the 2 types. Braverman[30] quoted an 8·2% mortality in 73 patients under the age of 20. Kjellberg[31] described 1 death at 14 years of age in his 15 patients.

TABLE I. MORTALITY RATE IN SIX SERIES OF CASES

Author	No. of cases	Mortality %
Braverman[30]	73	8·2
Nadas[17]	67	7·5*
Campbell[1]	40	5·0
Downing[26]	37	8·0
Marquis and Logan[25]	28	17·8**
Kjellberg[31]	15	6·6

* Over a 5-year period.
** Followed for 4 years.

Marquis and Logan[25] had 5 deaths in their 28 patients followed for 4 years. They, however, stated that 'sub-aortic stenosis as an isolated lesion appears to be very uncommon and rarely of sufficient severity to require operative treatment'. Downing[26] summed up the position thus: 'This is the only congenital heart disease compatible with a relatively long life that requires restriction of activity. Aortic stenosis constantly threatens acute coronary insufficiency. The margin between adequate and inadequate coronary flow is small and any activity may quickly exhaust the reserve and lead to failure and sudden death'. He reported 3 sudden deaths (from either sudden diminution in left ventricular output or carotid sinus reflexes) in 37 cases.

Symptomatology (Fig. 1)

Most patients are asymptomatic during infancy and childhood, but develop symptoms in adolescence. Rarely, marked cardiac failure occurs in infancy and sweats are a pronounced symptom. According to Marquis and Logan[25] even slight disability is significant. Fatigue is a common and early symptom, is often associated with palpitations and an awareness of forceful heart action, and is related to inadequate systemic blood flow. These symptoms indicate that the myocardium is beginning to feel the effects of an unyielding obstruction at the aortic-valve level.

Angina results from the discrepancy between coronary flow and myocardial oxygen demands. Of Campbell's[1] 40 patients, 11 suffered from angina, but all started complaining in their

teens. His youngest patient with angina was 13 years old. Of Downing's[26] 37 patients, only 1 had angina. Marquis and Logan[25] stated that angina usually occurs in older patients; it presented in only 1 of their patients, aged 24 years. One of the youngest recorded patients with angina was case 4 of Spencer,[15] who developed angina at 4 years of age. Thirteen of our patients complained of angina or some form of chest pain, not necessarily related to exercise — the youngest was a boy of 5 years of age (case 7). In case 2 — a girl of 17 — angina was incapacitating. Dizziness, probably from relative cerebral ischaemia, is a common symptom and occurred in 20% of Campbell's[1] 40 cases. Cardiac syncope is ominous, and again is related to inability of the left ventricle to increase cardiac output and cerebral blood flow during exercise.

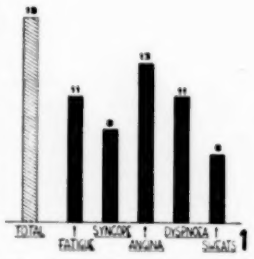

Fig. 1. Number of patients showing various symptoms (present series).

The increased requirements of the left ventricle can be met only by higher ventricular filling pressure, which is associated with elevated pulmonary venous pressure resulting in left ventricular failure and its symptoms, Dyspnoea usually occurs over the age of 30.

Physical Signs

1. General development is satisfactory except in those patients with other associated anomalies.

2. The pulse is often normal, sometimes diminished in amplitude, and occasionally virtually absent. Marquis and Logan[25] stated that the aortic orifice has to be reduced to less than a quarter of its normal size before the form of the arterial pulse is altered.

3. Blood pressure is often within normal limits. With associated aortic incompetence or coarctation of the aorta, it is abnormal.

4. There is a left ventricular lift to the apex beat which is forceful.

5. There is a palpable coarse systolic thrill over the aortic area, best felt on expiration and on leaning forwards. This thrill is felt over the carotids and in the suprasternal notch.

6. There is a loud harsh systolic stenotic murmur, best heard at the second right interspace, with wide transmission to the neck, back and chest. In very young infants it is stated by Spencer[15] that the murmur is best heard to the left of the sternum.

7. There is often an early systolic ejection click and the first sound is often loud and split.

8. The second heart sound over the aorta may be diminished or absent.

9. The first sound at the apex is often accentuated.

10. There is rarely an apical mid-diastolic murmur which disappears after successful valvotomy.

11. There is often (perhaps in 20 - 30% of cases) an early diastolic murmur along the left sternal border indicative of some aortic incompetence caused by dilatation of the ring from post-stenotic dilatation.

In our series of 18 cases, all 18 had a systolic murmur and thrill, 13 had a forceful left ventricular apex beat, and 4 had an early diastolic murmur.

Electrocardiography

The electrocardiogram is normal in most asymptomatic cases. The presence of left ventricular hypertrophy is always associated with moderate to severe outflow-tract obstruction according to Morrow.[22] In the 37 cases recorded by Downing,[26] 22 showed left ventricular hypertrophy.

Radiological Examination (Fig. 2)

The heart is often of normal size in asymptomatic cases. There is usually post-stenotic dilatation of the ascending aorta, best seen as an anterior prominence in the left anterior oblique view. Calcification in the ring or aortic cusps is exceptional under the age of 40. There is occasionally some prominence of the left atrium, but the pulmonary vasculature is invariably normal in the uncomplicated case. In the presence of associated lesions the radiograph may show other signs — e.g. notching of the ribs in coarctation.

Haemodynamic Studies

Brock and his associates[23] have shown the safety of transventricular left-heart catheterization. A needle is inserted through the anterior chest wall into the left ventricle. A polythene catheter is threaded through the needle and is passed up into the aorta. A withdrawal tracing may help to differentiate between valvular and sub-valvular stenosis. We have found this easy and safe in cases of acquired aortic stenosis, but we have had great difficulty in finding the left ventricular cavity in cases of congenital aortic stenosis, even at open operation. This we have shown to be caused by the marked concentric hypertrophy of the left ventricle, which leaves a minute chamber. When the heart is enlarged the procedure is simple, since the cardiac enlargement indicates cardiac dilatation and an enlarged chamber. This procedure can also be carried out transbronchially, but in the absence of an enlarged left atrium and in young children it is more dangerous. We have, therefore, performed few pre-operative left-heart catheterizations in this series, but have taken pressures at cardiotomy where indicated.

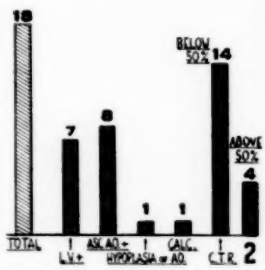

Fig. 2. Radiological signs found in this series. (LV + = enlarged left ventricle. Asc. Ao. + = dilatation of the ascending aorta. calc. = calcification. CTR = cardiothoracic ratio.

When hypothermia was used, because of the limitations of operating time, the distinction between valvular and subvalvular stenosis was of paramount importance. However, now that we use the pump oxygenator, the differentiation is academic, since there is ample time, not only for full assessment, but for reparative surgery. Spencer[18] agreed with this opinion, saying: 'left-heart catheterization is not advised as a routine', because the clinical diagnosis was often clear and the electrocardiogram was a good guide to the severity of the stenosis. Morrow[22] agreed that left-heart catheterization is the most helpful procedure for the evaluation of congenital aortic stenosis. However, for real value, not only the pressure, but also the flow across the valve, must be measured.

Angiocardiography

Selective left-heart angiocardiography will show the stenosis very beautifully, as demonstrated by a case reported by Bjorck.[24] We have not used this method in our cases.

Indication for Operation

Until a few years ago the prognosis was thought to be uniformly good and, in any case, no adequate surgery was available. Although we had used hypothermia, its limitations in this field were such that we operated upon urgent cases only. Now with dry-heart visual surgery with the pump oxygenator, the need for surgery can be matched with safety and adequacy in technique.

We advise surgery in all symptomatic cases and in all cases where there is left ventricular hypertrophy on electrocardiogram or on radiological investigation. Morrow[22] advised operation in all symptomatic cases and in asymptomatic cases where the pressure gradient is in excess of 70 mm.Hg or where the orifice is less than 0·3 sq. cm. per sq. metre of body surface. Nadas[17] advised surgery if the valve area is less than 0·6 sq. cm., or where the gradient is above 40 mm.Hg. Braverman[30] stated that patients with syncope, easy fatiguability, or left ventricular hypertrophy should be referred for surgery. Spencer[15] and Downing[26] supported these criteria. Campbell[24] stated: 'The outlook for these congenital cases without operation is, however, worse and more uncertain than we thought at one time. Sudden death, sometimes with no

obvious warning or after attacks of syncope or angina is not rare'.

Marquis and Logan[25] assessed the severity of the lesion and the basis for surgery as follows:

1. By increased force of contraction, compensation is maintained and symptoms are minimal. The presence of symptoms suggests that the stenosis is severe and compensation inadequate.
2. Hypertrophy alone seldom leads to sufficient cardiac enlargement for clinical or radiological detection. Clinical enlargement of the heart is usually caused by dilatation, and its presence, when there is no associated aortic incompetence, suggests that hypertrophy alone has proved inadequate and that the stenosis must be severe.
3. There is no evidence that a congenitally stenosed orifice gets larger with the increasing demands of a growing child. With an orifice of fixed size, compensation that is adequate in early childhood may become inadequate as body growth proceeds. Stenosis that appears mild in early childhood may become severe before adult life is reached.

OPERATIVE CORRECTION

The surgical procedures available before safe surgical by-pass were: closed transventricular or transaortic valvotomy and open-vision correction under hypothermia.

A. Closed 'Blind' Methods

1. *Brock's Transventricular Approach*

Although this approach through the left ventricle was introduced by Bailey in 1952 for acquired aortic valvular stenosis, it was popularized by Sir Russell Brock, whose technique was safer and simpler. It was also used for congenital cases. Marquis and Logan[25] operated on 6 patients, 5 of whom had postoperative aortic incompetence. Downing[26] reported 19 cases with only 1 operative death and improvement in 14. We have not used this technique at all, since we believed that incompetence was produced in valvular stenosis, and that the obstruction was insufficiently removed in the sub-valvular cases. From what we have seen at open operations, this method has no place in our modern armamentarium, but is of historic interest only.

2. *Retrograde through the Aorta*

This was said to have the advantage that patients died of haemorrhage instead of ventricular fibrillation!! Its limitations are similar to those of the transventricular approach.

B. 'Open' Vision Methods

1. *Under Hypothermia*

This was a great advance, since the valve now could be clearly visualized, and adequate and controlled valvotomy performed. However, in our experience, the time is so limited that there is no margin of safety if anything untoward is found or occurs. This method was first successfully employed by Julian[9] in 1955 and by Brock on 26 January 1956. As recently as 1957 Brock[6] stated 'for non-calcified cases open aortic valvotomy under direct vision and with hypothermia is used'. Brock[2] lost case 5 (a girl aged 7 years from Johannesburg) whose aortotomy incision tore, and 12 minutes of occlusion were necessary for closure. Dye[35] had no mortality in 5 cases, of which 2 were sub-valvular. Morrow[19] had 2 deaths in 10 cases. Swan[36] had 3 deaths in 11 cases.

In 1957 and early 1958 we operated on 4 patients under hypothermia, using Swan's[36] technique as modified by Bedford, Sellors and Sellick.[37] The first patient was a woman of 43 (referred by Dr. S. J. Fleishman) who had an associated coarctation of the aorta, and who developed irreversible ventricular fibrillation after valvotomy. The second patient, a girl of 16, was referred by Dr. G. R. McLeish with intractable disabling angina. She also had primary amenorrhoea. An excellent valvotomy was obtained, but the lower end of the incision extended beyond the clamp. A further period of venous occlusion allowed control of the incision. She collapsed 3 hours after operation from reactionary haemorrhage. Autopsy confirmed a Turner's syndrome with ovarian agenesis, and haemorrhage from the aortic incision.

The third patient, a male of 32 (referred by Dr. N. Segal of the Cardiac Clinic of the Johannesburg General Hospital), known to have had a murmur all his life, had a very successful valvotomy. Operation confirmed grossly calcified valves. These 3 patients were all incapacitated. The fourth was a woman of 21 years, referred by Dr. B. van Lingen, with a forceful apex beat clinically and left ventricular hypertrophy on electrocardiogram. Aortotomy showed normal valve cusps with a sub-valvular diaphragm which was readily incised and dilated. Soon after closure of the chest a reactionary haemorrhage was suspected and the bilateral thoracotomy incision was re-opened. A bleeding point on the aorta was controlled and she recovered from this, but not without incident. She developed anuria which was successfully combated.

2. *Open Vision with Cardiac By-pass*

There is no doubt from our experience that this is the only safe and adequate method. Cooley[20] has operated upon 13 patients of whom 3 were under 6 months of age.

TABLE II. RESULTS IN FIVE SERIES OF CASES—OPEN AORTIC VALVOTOMY WITH THE PUMP OXYGENATOR

Author	*No. of cases*	*Operative mortality %*	*Sub-valvular*	*Aortic incompetence*
Spencer[18]	12	0·0	4	2
Cooley[20]	13	7·5		
Morrow[32]	3	33·3		
Gross[38]	16	12·5		
Adler and Fuller (present series) ..	14	0·0	4	4*

* Increased postoperatively in 2 patients.

We operated on 4 patients under hypothermia, of whom 2 died, while there were no deaths in our 14 patients operated on under open vision with cardiac by-pass (Table II).

OPEN AORTIC VALVOTOMY WITH CARDIAC BY-PASS

The particular variety of pump oxygenator is not as important as the efficiency of its operation, the prudence of the surgery, the maintenance of normal blood pressure and pH during operation, and postoperative supervision. We believe, however, that the disc oxygenator combined with de Bakey pump heads in a heart-lung machine of the Gross type[38] is slightly better than the others currently available.[45]

Pre-operative Treatment

The patient is admitted a week before surgery and is again fully assessed clinically and haematologically. Our patients are digitalized, given diuretics if necessary,

schooled in physiotherapy, and introduced to the 'special' nurses who will be in charge of them when they return from the theatre. Three days before the proposed operation the whole Unit meets to discuss every aspect of preoperative therapy and operative technique. Blood is also taken on this day for grouping, to exclude any rare group which might entail extra work for the blood transfusion service. Blood is again sent off the day before surgery for compatibility tests.

We have found no problem with blood collected the evening before surgery. The South African Blood Transfusion Service has never yet failed to supply us with our blood transfusion requirements. The details of this service have recently been reviewed by Shapiro.[39] A glucose drink is given, 3 - 4 hours before surgery, to obviate any hypoglycaemia which might be present because of enforced starvation and the low sugar content of stored blood.

The patient is premedicated, and cannulations for venous and arterial pressure readings are performed under local anaesthesia in adults and older children. In infants and young children we prefer having this done under general anaesthesia.

Incision

We now use a vertical sternal split which gives excellent access to the heart. This incision is preferred to the bilateral 'suitcase' incision which crosses from one axilla to the other. It takes much less time to open and to close the vertical split and, since it is much less traumatizing to the chest wall, the thoracotomy is virtually painless postoperatively. It also has the advantage of not opening either pleural cavity.

Cannulation

For our arterial return we now employ the right femoral artery, using metal cannulae made from tracheotomy tubes. These have the advantage of being curved, with a thin wall and a large lumen. For venous return we use McGill's portex endotracheal catheters, inserted through separate incisions in the wall of the right atrium, and controlled with purse strings passed through a short length of rubber tubing. The inferior catheter is passed from above downwards through the side of the atrial appendage into the inferior vena cava. The superior is passed from below upwards through a separate incision in the side wall of the atrium. In left-heart surgery we also pass a coronary sinus sucker through the tip of the left atrial appendage into the left atrium for decompression of the left ventricle; this is especially necessary after by-pass. The venous line is now fitted from the machine to the nylon 'Y' connection, care being taken not to capture any air bubbles which could act as an air lock and interfere with siphonage. Anoxic or cold arrest is now used in preference to the potassium arrest of earlier cases.

Phase of Partial By-pass

Immediately before going on to partial by-pass, a mixture of 98% oxygen and 2% CO_2 is fed into the oxygenator, and the anaesthetist prepares for further 'scoline', which is usually necessary because of dilution of the anaesthetic when by-pass is instituted. Every connection, as well as the additional sucker which has been attached to the coronary sinus sucker, and which will be used to keep the intracardiac operative field dry, is tested. Venous pressure, arterial pressure, and electrocardiogram are all checked. When the surgeon is satisfied on these points partial by-pass is instituted.

Phase of Total By-pass

The superior vena caval catheter is now introduced into the superior vena cava from its position in the right atrium. Momentary occlusion of the inferior vena caval catheter ensures patency and drainage of the superior catheter. When this has been shown to be satisfactory, the caval encircling ligatures are tightened and secured in position by clamping the ligatures distal to their rubber shrouds.

Phase of Total Cardiac Occlusion

After full by-pass has been established for several seconds, and when monitoring of arterial and venous pressure is satisfactory, the anaesthetist keeps the lungs inflated with helium, but does not inflate them rhythmically. A transverse sinus clamp is now placed across the aorta just below the innominate artery.

Open Cardiotomy Phase

The aorta is now incised with a No. 15 Bard-Parker blade and the incision is extended for 2 inches with a right-angled Pott's scissors. This extends down almost to the origin of the right coronary artery. Blood is now sucked away by the 'coronary sinus sucker' and the aorta and its valves are carefully inspected. Blunt nerve hooks are used to assess the valves and the stenosis. Again, a sharp scalpel blade is used to divide the fused commissures most carefully to within 2 mm. of the ring. In several of our earlier cases we cut only 2 commissures for fear of causing incompetence, in accordance with Spencer's[18] recommendations. This, we believe, is wrong, and we now carefully divide all 3 commissures. Six of our 18 cases have been bicuspid. At that time we were not aware of Austen, Shaw and Scannell's[40] work at the Massachusetts General Hospital where, with autopsy perfusion, they showed that 'division of only 2 of the commissures results in moderate aortic stenosis'.

After adequate valvotomy (aided if necessary, as in case 12, by gradual controlled stretching with a Brock's 3-bladed dilator), or if no valvular stenosis is found, the sub-valvular area of the outflow tract of the left ventricle is most carefully inspected for sub-valvular obstruction. In 3 of our 18 cases this was found. The diaphragm was either carefully incised and dilated, or partially excised and dilated. A satisfactory opening was accepted when an index finger and the largest Brock's bougie passed in readily.

Closure of Aortotomy

The left atrial suction is reduced sufficiently to allow blood to spill up from the left ventricle through the valves into the aorta. A finger is inserted down through the cusps into the left ventricle to empty it of any trapped air. The incision is then carefully closed with a running mattress suture. The first and last stitches are placed beyond the line of the incision and blood is allowed to fill the aorta before tying the last 2 stitches. When the aorta is filled, any retained air is aspirated by needle

puncture. Firm pressure is now applied to the aortotomy incision and the clamp across the aorta is slowly released. Coronary-artery flow now commences immediately and cardiac action gradually returns. In case 5 ventricular fibrillation appeared, but the heart defibrillated readily.

Cessation of By-pass

When cardiac action is seen to be effective, as judged by maintenance of arterial pressure, satisfactory electrocardiogram, and normal venous pressure, the caval ligatures are loosened, inflation of the lungs with 100% oxygen is commenced, and the superior vena caval catheter is withdrawn into the body of the right atrium. The inferior vena caval catheter is then completely withdrawn and its incision controlled by tying the purse-string suture. Left atrial suction is still being gently employed to assist the left ventricle. After a few minutes, when blood balances have been carefully assessed, by-pass is stopped, but the remaining superior vena caval catheter is withdrawn only when the electrocardiogram and arterial pressures are completely satisfactory. Immediately thereafter our attending haematologist gives the calculated dose of polybrene (previously protamine sulphate) slowly, to reverse the heparin. Pressures are now taken again in the left ventricle and from the central arterial pressure line to see if the gradient has been abolished.

Closure

The left atrial catheter is now withdrawn. All sutures are carefully inspected, catheters to drain the pericardium and mediastinum are placed, and the anterior pericardium loosely approximated. In cases operated on subsequent to this initial series, the pericardium has been widely fenestrated into the right pleural cavity, which has been drained by a large underwater seal catheter. The sternum is held together firmly by wiring, and the incision carefully closed in layers. The patient is X-rayed at the end of the operation, in the theatre, and bronchoscopy performed if necessary. In case 11 an unsuspected left pneumothorax was demonstrated and decompressed.

Medical and Biochemical Control

Dr. M. Zion and Dr. L. Braudo have been responsible for constant monitoring of the arterial pressure and electrocardiogram during operation, and have personally supervised the early postoperative care. Mr. L. du Plessis, later Mr. D. Evans and Mr. W. du Plessis, have managed the pump oxygenator and have been guided by the arterial pressure (we believe cerebral circulation is adequate in the presence of a satisfactory systemic pressure), the venous pressure and the venous and arterial oxygen saturation. Drs. H. B. W. Greig and A. Walker of the South African Institute for Medical Research have, with their mobile laboratory attached to the theatre, estimated heparin and protamine (later polybrene) requirements, and monitored both venous and arterial oxygen saturations. Their frequent pH estimations during operation and especially after by-pass, and in the critical 8 hours following operation, have been invaluable. They have also performed a large number of haematological and biochemical estimations which have confirmed the safety of the Gross type of pump oxygenator.

Dr. W. Scott has made fluid balance his concern and has been responsible for simple venous monitoring, a valuable index of blood depletion or of overloading.

Postoperative Complications (Table III)

A. *Special to perfusion cases:*

1. Post-perfusion syndrome with hypotension, peripheral cyanosis and irritability. This is not common where high flow rates have been employed and oxygenation has been satisfactory; it has not been seen in this series. However, case 5 showed marked postoperative restlessness.

TABLE III. OPERATIVE AND POSTOPERATIVE COMPLICATIONS IN PRESENT SERIES

Case no.	*Complication*
4	Reactionary haemorrhage
4	Aggravation of aortic incompetence (subvalvular)
5	Ventricular fibrillation
7, 9, 11 and 12	Post-pericardiotomy syndrome
8	Cerebral oedema
10	Mild wound disruption
11	Aggravation of aortic incompetence
7	Mild aortic incompetence induced

Case 8 became comatose with elevated blood pressure 36 hours postoperatively, and a lumbar puncture showed a pressure of 300 mm. H_2O, relieved by slow decompression.

2. Immediate acidosis is usually caused by inadequate perfusion with a low mean pressure. Delayed acidosis is usually caused by inadequate postoperative ventilation following pain, pleural haemorrhage or bronchial secretions.

3. Reactionary haemorrhage, of sufficient degree to necessitate thoracotomy, has not occurred in our pump cases. The patient, case 4, operated on under hypothermia, was re-opened while being re-warmed.

4. Pulmonary complications are much less common now that we employ the sternal split, though case 8 had a left-lower-lobe atelectasis. The aetiology and prevention of chest complications has been fully described by Kolff and Effler.[42]

5. Thirst is a most distressing symptom, both for patient and attending staff. It is caused by hyperosmolarity of the blood and is not assuaged by drinking fluids. Fluid control has been very carefully supervised. This has been fully reviewed by Sturtz *et al.*[42] from the Mayo Clinic and du Plessis and Scott[43] from early experience with our patients.

6. Transient cardiac failure when arrest has been prolonged can occur, but, in our series, was only seen in case 7.

7. Prolonged postoperative pyrexia, as in the post-commissurotomy syndrome described by Papp and Zion,[44] occurred in cases 7, 9, 11 and 12.

8. Septicaemia from infection during by-pass has been described, but fortunately we have not yet encountered this dread complication.

9. Postoperative haematological changes after a few days are minimal — mild anaemia and leucocytosis. (In case 13 two transfusions were necessary 14 days postoperatively.)

B. *Normal postoperative complications* common to any major transthoracic procedure.

Postoperative Results

1. The systolic murmur decreased, but never vanished.

2. The pressure gradient was diminished in all, but not often abolished except in case 8.

3. Mild aortic incompetence. We produced this in case 7 and aggravated a pre-existent aortic incompetence in case 11.

4. All patients seen postoperatively were relieved of their symptoms. Case 7, aged 5, with disabling angina, has been relieved completely and is very active and well.

These operations of necessity required team-work, and we should like to thank our many medical colleagues, the technicians, and the nursing staff, who all formed part of the team. Also, we thank those colleagues who referred the patients to us for operation.

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A CLINICAL CLASSIFICATION OF CEPHALO-PELVIC DISPROPORTION

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'The art of obstetrics is more often travestied in the name of disproportion than in any other instance. The prediction that cephalo-pelvic disproportion will adversely influence labour, as well as the assessment of its extent and the treatment with which the problem is met, are more than matters of mathematical measurement and demand a skill and experience which no textbook can provide.' (Donald, 1959.)

Many vaginal deliveries, after Caesarean section in an earlier pregnancy, occur in patients in whom the original indication for the operation was disproportion. A number of these subsequent infants are larger than the firstborn. As a result, doubt exists whether the original indication was genuine, or whether the findings at the time of the operation were correctly interpreted. The looseness of the very term 'disproportion' may account for some of this inexactitude. An attempt has therefore been made to classify disproportion as a clinical presentation. This classification will not in any way detract from the skill required in the assessment and management of such cases. Nevertheless, if it is applied retrospectively as a routine when making the final summary of a particular labour, it will aid the obstetrician in the conduct of subsequent pregnancies and labours.

ULTIMATE TREATMENT OF CEPHALO-PELVIC DISPROPORTION

Clinically, the ultimate treatment of cephalo-pelvic disproportion may be listed under 6 main headings. In some instances 2 or more of these groups overlap:

1. Caesarean section — an operation to bypass the obstruction that exists between the foetal head and the maternal pelvis.

2. Craniotomy or tapping of the cerebrospinal fluid — operations that reduce the size of the foetal head.

3. Symphysiotomy or pubiotomy — operations that increase the capacity of the maternal pelvis.

4. Manoeuvres designed to alter the presenting diameter of the foetal head to a more favourable size, or even to change the presentation itself to a breech, e.g. Thorn's manoeuvre and internal version.

5. Procedures which aid descent, or both descent and rotation of the vertex, e.g. forceps delivery, and rotation and delivery using Kielland's forceps.

6. Procedures adopted to improve the efficiency of uterine action where such action is hypotonic or incoordinate, e.g. oxytocic-drip infusions, continuous caudal and epidural anaesthesia, and the use of the 'vacuum extractor' before full dilatation of the cervix.

ANALYSIS OF THE CAUSES OF CEPHALO-PELVIC DISPROPORTION

When analysed, the treatments employed in groups 1-3 indicate that an absolute obstruction to vaginal delivery exists *throughout* the labour. The disproportion is not related to the presenting diameter of the foetal head. In the majority of such cases the diagnosis is confirmed before the onset of labour. The cause for the cephalo-pelvic disproportion is in the maternal pelvis or in the size of the foetal head. If it is in the maternal pelvis,

e.g. contracted pelvis, it is permanent and will operate in all subsequent labours. If it is in the size of the foetal skull, e.g. hydrocephalus, the cause is temporary and, therefore, unlikely to recur in further confinements.

The procedures employed in groups 4 - 6, when successful, achieve vaginal delivery of a living infant without change in the size of the maternal pelvis. The cephalo-pelvic disproportion that exists in such cases is relative. It may be suspected before the onset, but is rarely confirmed until the patient has been in labour for many hours. Factors which may lead to or are associated with relative disproportion are:

(*a*) A presenting diameter of the foetal head other than the suboccipito-bregmatic or submento-bregmatic (mento-anterior).

(*b*) A presentation, such as occipito-posterior, which could complicate the mechanism of labour.

(*c*) A failure of progressive dilatation of the cervix. This finding may be either the cause or the effect of an unfavourable presentation or position.

It is accepted that many of the patients managed by the techniques of groups 4 - 6 are ultimately delivered by Caesarean section. This is particularly so where there is an additional factor, such as prolonged labour or foetal distress. It is following upon Caesarean sections in this category of disproportion that vaginal delivery of larger infants occurs. Therefore, the indication for the operation must not be classed as disproportion unless a large infant is delivered or a postpartum X-ray pelvimetry reveals a major degree of contracted pelvis.

CLASSIFICATION

A clinical classification of cephalo-pelvic disproportion, based on the above observations, can thus be formulated, as shown in Table I.

An obvious fault of this classification is that it is based mainly on the assessment of cases as they appear in labour. This is not as great a drawback as it may at first seem. In cephalo-pelvic disproportion the problem of management often only arises once the patient is in labour. Also, the conduct of subsequent pregnancies and confinements is based on a retrospective analysis of the findings and outcome of the initial labour.

TABLE I. CLINICAL CLASSIFICATION

Cephalo-pelvic disproportion

Absolute (*true mechanical obstruction*)		*Relative*
Permanent (*maternal*)	*Temporary* (*foetal*)	
Contracted pelvis	Hydrocephalus	Brow presentation
Pelvic exostoses	Large infant	Face presentation — mento-posterior
Spondylolisthesis		Occipito-posterior positions
Anterior sacro-coccygeal tumours		Vertex presentations with a diameter other than the suboccipito-bregmatic presenting. The so-called 'deflexed head'

The unqualified use of the term disproportion as an indication for any obstetric procedure is not sufficient nor justified. It should always be coupled with the underlying cause and with a clear statement as to the state of the pelvis, e.g. absolute disproportion — contracted pelvis; relative disproportion — hydrocephalus, pelvis normal; or relative disproportion — occipito-posterior position, pelvis with an android brim.

In this manner much that is speculative in the management of labour following Caesarean section will be eliminated.

This view was probably held by those who formed the Standard Maternity Hospital Report Committee of the Royal College of Obstetricians and Gynaecologists. In the section covering disproportion, the cause (including any pelvic abnormalities) is requested.

SUMMARY

1. The ultimate methods of treatment in the management of cephalo-pelvic disproportion are analysed.

2. Based on this analysis a clinical classification of cephalo-pelvic disproportion has been formulated.

3. It is submitted that this classification will aid the obstetrician in the management of subsequent pregnancies and labours.

MEDICAL ASSOCIATION OF SOUTH AFRICA : MEDIESE VERENIGING VAN SUID-AFRIKA

ANNUAL GENERAL MEETING

The Annual General Meeting of the Medical Association of South Africa was held at the Medical School, Cape Town, on 22 September 1961, when Mr. J. A. Currie was inducted as President of the Medical Association of South Africa by Dr. W. Chapman, the retiring President. Dr. Chapman referred to the services which Mr. Currie has rendered to the Association, the medical profession, and the public. In replying Mr. Currie expressed his thanks and appreciation, and he thanked Dr. Chapman for the way in which he had discharged his duties during his term of office.

After dealing with some more formal matters, the meeting adjourned until Monday 25 September 1961.

THE ADJOURNED ANNUAL GENERAL MEETING AND MEDICAL CONGRESS

The adjourned Annual General Meeting of the Medical Association of South Africa was held in the City Hall, Cape Town, on 25 September 1961 at 8 p.m. A large audience of members and their wives, together with overseas guests who attended the Congress, was present.

Opening of Congress

The platform party consisted of the following: Mr. J. A. Currie, President of the Medical Association of South Africa, and Mrs. Currie; Councillor A. H. Honikman, Mayor of Cape Town, and Mrs. Honikman; Mr. J. P. Duminy, Principal and Vice-Chancellor of the University of Cape Town, and Mrs. Duminy; Dr. E. W. Turton, Chairman of Federal Council, and Mrs. Turton; Dr. W. Chapman, Retiring President of the Medical Association, and Mrs. Chapman; Dr. J. C. Coetzee, Vice-President of the Medical Association; Dr. A. Goldberg, President of the Cape Western Branch of the Medical Association; Dr. A. H. Tonkin, Secretary of the Medical Association of South Africa; the Rev. John Aubrey, the President's Chaplain; and Dr. A. Mears, Secretary, Cape Western Branch of the Medical Association and mace bearer.

After the entry of the platform party, the Rev. John Aubrey, the President's Chaplain, led the meeting in prayer. His Wor-

ship the Mayor of Cape Town, Councillor A. H. Honikman, welcomed the members of Congress, and Dr. E. W. Turton replied suitably. The President then introduced Mr. J. P. Duminy, Principal and Vice-Chancellor of the University of Cape Town, and invited him to open Congress.

In addressing Congress on the subject of 'The romance and humanism of medicine', Mr. Duminy, *inter alia*, said:

> 'Never in the history of the world has mankind stood in greater need of men of goodwill, of vision, and of fearlessness. The world today presents a puzzling and an awesome picture: it has been reduced by the marvels of science and technology to the merest unit in which all men now are neighbours — and the change has taken place at such great speed that mankind has not had the time to adapt its thinking, to readjust its attitudes, and to attune its actions to situations which call for close collaboration and common planning if we are to find a way in which all can still have a share in the good things that the world has to give.
>
> 'We are neighbours, and yet we are strangers and enemies. This cruel paradox has led to a situation which is becoming progressively more dangerous before our very eyes — and the sands of time are running out.'

Mr. Duminy concluded his address by saying:

> 'You men of medicine have always been in the vanguard of the crusade in keeping mankind ever mindful of those fundamental human issues, which I believe to be the most worthy and most demanding of our time and our attention. We look to you, and we look to you with confidence, to use the ample power of your influence in the struggle to bring the world back to a proper realization of the essential meaning of existence, of the sacredness of life, and of the dignity of Man.'

The Secretary of the Association, Dr. A. H. Tonkin, then presented the distinguished overseas visitors to the President.

Presentation of Awards

Mr. J. A. Currie presented the insignia of Immediate Past President to Dr. Chapman and Mrs. Currie, the President's lady, was presented with her badge of office by Mrs. Chapman.

Gold medal. The Secretary read a citation in respect of Dr. J. H. Struthers to whom the award of the Association's Gold Medal was made for distinguished services to the profession.

Silver medal. The Secretary read a citation in respect of Prof. J. F. Brock, whom the Association desired to honour for his distinguished services to medical science and humanity.

Bronze medals. The awards of the Association's Bronze Medals were made to Drs. Alan Taylor of Durban, L. M. Marchand of Pretoria (Associate Secretary of the Medical Association), and Dr. E. W. Turton, Chairman of Federal Council.

The Hamilton-Maynard Memorial Medal for the year 1959 was awarded to Prof. Jannie Louw in recognition of his article: 'Malformations of the anus and rectum' which was published on p. 874 of the *Journal* for 17 October 1959.

The Hamilton-Maynard Memorial Medal for 1960 was awarded to Prof. C. E. Lewer Allen in recognition of his article: 'The University of Cape Town artificial limb', which was published on p. 125 of the *Journal* for 13 February 1960.

Presidential Address

Mr. Currie then delivered his Presidential address. The address was published on p. 845 of the issue of the *Journal* for 14 October 1961.

After the President had delivered his Presidential Address, a Mayoral reception was held in the Old Drill Hall.

FEDERAL COUNCIL MEETING

A meeting of the Federal Council of the Medical Association was held in the week preceding Congress, on 21 - 23 September 1961, in the Bacteriology Lecture Theatre, Medical School, University of Cape Town. The Chairman of Federal Council, Dr. E. W. Turton, presided. Among those present were the President of the Association, Mr. J. A. Currie; the Past President, Dr. W. Chapman; and the Honorary Treasurer, Mr. J. D. Joubert.

Among the matters discussed was the problem of the Association's policy regarding insurance companies that operate in the field of prepaid medical insurance.

Federal Council considered certain recommendations by the Central Committee for Contract Practice in connection with the present agreements between the Association and SANSOM and between SANSOM and its members. Federal Council also interviewed Dr. H. Hansmann, Managing Director of SANSOM.

After prolonged discussion, during which all aspects of these agreements were taken into consideration, Federal Council resolved to direct its Central Committee for Contract Practice to conduct further negotiations with SANSOM with a view to effecting certain amendments to the existing agreements.

The full minutes of this meeting of the Federal Council will be published in an early issue of the *Journal*.

THE MEDICAL CONGRESS

The records show that 990 members of the Medical Association registered and attended the 43rd South African Medical Congress held in Cape Town from 24 to 30 September. Among the distinguished visitors from overseas who attended the Congress were: Dr. Ian D. Grant, Chairman of the British Medical Association; Dr. D. P. Stevenson, Secretary of the British Medical Association; and Dr. H. Hethrington, representative of the Canadian Medical Association.

Further reports on the proceedings of the Congress will be published in an early issue of the *Journal*, and some of the papers delivered at Congress will be published in the *Journal* in due course.

MEETING OF MEMBERS OF FEDERAL COUNCIL WITH REPRESENTATIVES OF THE BRITISH MEDICAL ASSOCIATION AND THE CANADIAN MEDICAL ASSOCIATION

On Monday 25 September, an informal meeting was held between members of the Federal Council present at the Congress of the Medical Association, and Dr. Ian Grant, Chairman of the Council of the British Medical Association; Dr. D. P. Stevenson, Secretary of the British Medical Association; and Dr. H. Hethrington, representative of the Canadian Medical Association.

Dr. E. W. Turton, Chairman of Federal Council, asked these doctors to give the members information on matters of common concern to the three Associations. Dr. Grant stated that this year the subscription of the BMA had been increased from £6.6.0 to £9.9.0 annually (it was £3.3.0 annually in 1945). Although this subscription was relatively high, there was a sliding scale covering the first nine years after qualification, and there was a reduction for university teachers. Branches of the BMA received a 6s. 8d. capitation fee for their members throughout Great Britain. There are now about 50,000 members of the BMA, being 80% of the total number of doctors in the country.

Discussing the National Health Service in Great Britain, Dr. Grant said that it worked well on the whole, although there was much criticism of the fact that there was no incentive to the general practitioner to do better work — he received the same capitation fee for the patients on his list whether he had just qualified or had been in practice for many years. Consultants were, on the whole, better off, and one in three were given merit awards, going up to £4,000 a year. These awards are not publicized since it was felt that the publication of the names of the recipients would constitute a form of advertising. The maximum list of a general practitioner was 3,500 patients, and it had been estimated that each patient received an average of 5.5 services from his doctor a year, so that it could be seen that a doctor with a full list was kept extremely busy. Only about 2% of patients transferred from one doctor's list to that of another practitioner.

The National Health Service cost £670,000,000 a year at present, and this was only for running the service. It did not

take into account any capital expenditure, and, in fact, until the last few years, no new hospitals had been built in Great Britain since the inception of the Service in 1948. Although the Service cost the taxpayer this amount of money, it was indirect payment for his medical services, and most patients in the country were satisfied with the arrangements. About 5% of the population still preferred to have private attention, and many belonged to one of three insurance schemes for private medical attention, all sponsored by doctors.

Dr. Stevenson said that there was a shortage of doctors in Great Britain, and that, on the average, 40% of the staffs of hospitals were overseas graduates. Although three new universities had been founded since 1945, no new medical faculties had been instituted.

Dr. Hethrington stated that private enterprise was still the accepted method of medical practice in Canada, but there was a Royal Commission sitting in Ottawa at the moment reviewing all aspects of the financial side of medical practice. Every Province in Canada had a form of prepaid medical insurance, and this seemed to be working satisfactorily.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during August 1961 are gratefully acknowledged:

Met dank word die volgende skenkings gedurende die maand Augustus 1961 erken:

Votive cards in Memory of:Geloftekaarte ter Nagedagtenis aan:

Mr. Cowie *by* Natal Inland Branch M.A.S.A. and Dr. F. C. Friedlander; Dr. L. Jacobson *by* Dr. and Mrs. R. Morris and daughter, Dr. A. Segil, Dr. B. M. Burger, Drs. G. Nienaber and E. W. Turton, Drs. A. D. and B. Polonsky, Drs. Myer, Noll and Klaff; Mrs. Ruby Linder *by* Dr. V. Brink; Dr. J. B. Mitton *by* Dr. A. W. Sichel; Dr. C. Becker *by* Dr. R. J. Fleming, Dr. J. W. Schabort, Transvaal Branch of South African Society of Anaesthetists; Mr. S. R. Clarke *by* Dr. V. Brink; Mrs. Henry *by* Dr. R. B. Peckham; Mev. Ferreira *deur* Mev. J. S. du Toit en Anton.

Total Received from Votive Cards: / *Totaal Ontvang van Geloftekaarte:* } R92.65

Services Rendered to: Dienste Gelewer aan:

Mr. Ben Fine *by* Mr. S. Scher.
Dr. E. Schultz *by* Dr. C. Michelow.
Neil, son of Dr. J. K. McKechnie, *by* Drs. Jackson and N. Laubuschagne.
Eugeni, daughter of Dr. S. B. Lange, *by* Drs. L. Lane and P. B. Hafner.
Dr. D. R. Morris *by* Drs. Sims, Gluckman, Bloomberg and Holman.
Mrs. Kramer *by* Mr. H. M. Pretorius and Dr. R. J. P. Venning.
Derek Kramer *by* Dr. F. Walt, Dr. F. Stern, Drs. Drummond, Rifkin and Roach.
Dr. D. Frost *by* Mr. T. V. Simpson.
Mrs. B. W. Franklin Bishop *by* Mr. H. Pretorius, Mr. M. Stein, Dr. F. Davidson, Dr. L. E. Wolpert, Drs. Drummond, Duncan Taylor and Rifkin.

Total Received from Services Rendered: / *Totaal Ontvang van Dienste Gelewer:* } R157.75

Donations: Skenkings:

Drs. Harries, Kuschke, Hofmeyr, Justin van Selm, G. Comay, M. J. Cohen, A. L. Forbes, T. Pearlman, K. H. Dyke, J. H. Botha, J. J. Lawrence and G. L. St. J. Whittington-Winford.

Cape Western Branch M.A.S.A. (Collection Box)	8.20
Dr. M. Byala	6.30

Total Received from Donations: / *Totaal Ontvang van Skenkings:* } R26.90

Grand Total : Groot Totaal: R277.30

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Nico van der Merwe has been appointed Chief Medical Inspector of Schools for the Cape Province, as from 1 October 1961.

Dr. Nico van der Merwe is aangestel as Hoof Mediese Inspekteur van Skole in die Kaapprovinsie vanaf 1 Oktober 1961.

* * *

Dr. I. P. Jaffe, paediatrician, of Cape Town, has returned from an overseas study tour and has resumed his practice in partnership with Drs. Shore and Rabkin.

* * *

Dr. J. M. van Niekerk, radiologist, of Pretoria, has commenced practice in partnership with Drs. Wolpowitz, van Rhyn and Lundie at Robert Koch Medical Building, and at the Medical Centre, Pretorius Street, Pretoria. Telephones: Rooms 24512, residence 78-4705.

Dr. J. M. van Niekerk, radioloog, van Pretoria, het begin praktiseer in vennootskap met drs. Wolpowitz, van Rhyn en Lundie, in Robert Koch Mediese-gebou en in die Mediese Sentrum, Pretoriusstraat, Pretoria. Telefone: Kamers 24512, woning 78-4705.

* * *

Society for Endocrinology, Diabetes and Metabolism, Natal Branch. The next meeting of this Society has been postponed to Monday 13 November owing to the television programmes being shown. The meeting will then be held in the Nurses' Lecture Theatre, St. Augustine's Hospital, at 5 p.m. Tea will be served at 4.30 p.m. A discussion on the applied physiology of the thyroid gland will be led by a physician, a surgeon and an isotope worker, and will be entitled 'Why does anybody use Lugol's iodine?' All those interested are welcome to attend this meeting.

* * *

The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 30 October 1961 at 5.10 p.m. in the Institute Lecture Theatre. The speakers will be Drs. A. H. Rubenstein and N. W. Levin, and the topic will be 'A case of diabetes mellitus treated with manganese; and the physiology of manganese'.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 25 October at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. The speakers will be Drs. D. Harwood-Nash and A. Hudson, and the topic will be 'Neonatal sepsis'.

* * *

Dr. John D. L. Hansen has been promoted to associate professor in the Department of Child Health at the University of Cape Town Medical School. Dr. Hansen, who has done extensive research on malnutrition in children, joined the Department in 1955 as a lecturer. In 1950 he was awarded the Cecil John Adams Travelling Scholarship and spent 2½ years at paediatric centres in the United States; and in 1960 he was awarded a Rockefeller Travelling Scholarship.

* * *

Dr. Christiaan Neethling Barnard, who is in charge of surgical research at the University of Cape Town, has been promoted to associate professor in the Department of General Surgery.

Dr. Barnard, who is an authority on open-heart surgery, is at present overseas on a lecture and study tour and will return to South Africa in about six weeks. In 1960 Dr. Barnard was awarded the Ernest Oppenheimer Memorial Trust Bursary and visited Russia, the United States and Britain.

* * *

Dr. C. C. Pretorius, verloskundige en ginekoloog, van Pretoria, het onlangs sy praktyk hervat na 'n reis in Europa waar hy o.a. die Internasionale Kongres vir Sitologie en Verloskunde en Ginekologie in Wenen bygewoon het. Dr. Pretorius het besoek afgelê by klinieke in Wenen, Stockholm, Spanje, en Holland.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING SEPTEMBER 1961

12th Middle East Medical Assembly, Beirut, 4 - 6 May 1962. American University, Beirut, Lebanon.

3rd International Congress of Infectious Pathology, Hamburg, May 1962. Prof. H. Pette, President, Martinstr. 52, Hamburg 20, Germany.

2nd Asiatic Congress of Obstetrics and Gynaecology, Calcutta, 23 - 25 January 1962. Chairman, Subodh Mitra, 4 Chowringhee Terrace, Calcutta 20, India.

International Anaesthesia Research Society, 36th Congress, Bal Harbour, Florida, 18 - 22 March 1962. Dr. A. William Friend, 227 Wade Park Manor, Cleveland 6, Ohio.

International Neurological Meeting, Paris, 5-6 June 1962. Dr. Jean Sigwald, Secrétair Général, 68, boul de Courcelles, Paris 17e, France.

BOOK REVIEWS : BOEKBESPREKINGS

YEAR BOOK OF DERMATOLOGY

The Year Book of Dermatology (1960 - 1961 Year Book Series). Ed. by Rudolf L. Baer, M.D. and Victor H. Witten, M.D. Pp. 478. Illustrated. $9.00. Chicago: Year Book Publishers. 1961.

The latest *Year Book of Dermatology* presents, as usual, abstracts of the best of the American literature and a sprinkling of news from the world outside. Schmid's (1960) article on acquired cutaneous porphyria in Turkey is abstracted, although Cihad Çam's original observations (1957 onwards) were never noticed. Çam (*sic*) had to switch to French to make first base with his epidemic dermatosis of infants.

The editorial on drug eruptions is excellent and draws attention to the increase in incidence of reactions of photosensitivity. J.M.

OPHTHALMOLOGY

System of Ophthalmology. Edited by Sir Stewart Duke-Elder. Vol. II. The Anatomy of the Visual System. By Sir Stewart Duke-Elder, G.C.V.O., F.R.S. and Kenneth C. Wybar, B.Sc., M.D., Ch.M. F.R.C.S. Pp. xxii + 901. 842 illustrations and 3 coloured plates. R13.00. London: Henry Kimpton. 1961.

This second volume of Duke-Elder's *System of Ophthalmology*, which will replace the *Textbook of Ophthalmology*, deals with ophthalmic anatomy. Here anatomy and function are brilliantly interwoven.

True to the literary style of the 'old master', it chats anatomy with the reader. The subject is brought to life and becomes vitally interesting.

This volume covers ophthalmic anatomy in its entirety, both gross and microscopic, and their relation to function. The printing and colour illustrations are excellent. There is little reviewing necessary except to say that it maintains the high standard that the world has learnt to expect from Duke-Elder.

It will find its place in medical book-shelves and libraries all over the world without any prompting. M.H.L.

CORRESPONDENCE : BRIEWERUBRIEK

LIMITS FOR GENERAL PRACTITIONERS

To the Editor: Dr. Hamilton's letter in the *Journal* of 30 September[1] is interesting in that a case can be made out for anything if the premise one's arguments are based on is in the first place false.

It surely must be the claim of very few general practitioners that they can deal more efficiently with 'head injuries, penetrating wounds of the chest, the many and varied bony injuries, the multitudinous female complaints and the "bits and pieces" of the general surgeon' than specialists in these fields. They can however claim that they can deal with these conditions better than any other person on the spot, and if the case happens to be urgent they are in duty bound to deal with it.

I think what the South African Medical Council is referring to is not the above type of case, but the patient who, for example, has been shown to have gallstones with dyspepsia and occasional attacks of colic, or the patient with a thyroid requiring operation, or with a chronic duodenal ulcer, who is sick of conservative treatment, or the patient with the easily reducible hernia who can travel quite comfortably in one afternoon to seek the services of someone trained in the art of surgery.

To the public the doctor is still a very important man — one might almost say an omniscient man — and one can hardly expect the public to know the difference between M.B., Ch.B. or M.R.C.P. or F.R.C.S. or M.R.C.O.G.

I know that this very same public will rather belittle the doctor who refuses to do the operation when the fellow next door is doing it. Has this point perhaps not coloured Dr. Hamilton's view? I also know that there are many senior general practitioners who are concerned about 'fools rushing in where angels fear to tread'.

Dr. Hamilton's last point that 'surely the correct approach to the problem is to educate *all* doctors into a realization of their own capabilities and limitations' has already been dealt with. The doctors know full well what their capabilities and limitations are. It is the public that does not.

J. R. Frylinck
Surgeon
403 Lister Buildings
Jeppe Street
Johannesburg
4 October 1961

1. Correspondence (1961): S. Afr. Med. J., **35**, 824.

DIE PLATTELANDSE ALGEMENE PRAKTISYN

Aan die Redakteur: Ek wil my graag aansluit by wat dr. Hamilton in die jongste uitgawe van u *Tydskrif* aangaande die waardigheid en status van die plattelandse algemene praktisyn geskryf het.[1]

Dit is duidelik dat die tyd aangebreek het waarin die gesaghebbende mediese instansies in ons land die verskillende

vorms van algemene praktyk uitken. Dit is noodsaaklik dat die plattelandse geneesheer wat uit eie beweging, aanleg, en ondernemingsgees daar praktiseer, onderskei moet word van sy stedelike, ongespesialiseerde kollega wat in minder gewaagde en derhalwe minder interessante mediese velde beweeg. Hierdie ongelykheid van vernuf veral ten opsigte van chirurgie, narkose, en verloskunde het die stedelike praktisyn oorval in die donker dae toe die konsulterende geneesheer plek moes maak vir die spesialis. Die engheid van laasgenoemde se spoor het die breër weg aan repe gesny. Hierdie verwikkeling het tot 'n nog pynliker mate die eens trotse algemene chirurg getref, wat vandag tussen die oorblyfsels: tiroïed, buik, spataar en maag-derm kanaal sy heil moet vind. Dit bly vir my 'n voortdurende bron van leedvermaak dat ginekoloë elke appendiks (wat nog daar is) tydens die histerektomie verwyder, terwyl geen algemene chirurg hom durf wreek deur die uterus (wat nog daar is) tydens 'n laparotomie te verwyder nie.

Dit kom my verder voor asof die meerderheid dosente 'n diepgaande wantroue in hulle mediese opleidingsvermoë openbaar. Byna by elke geleentheid waar die saak genoem kan word, moet ons hoor van die gebrekkige opleiding wat studente kry, en selfs die verpligte huisdokterskap word as onvoldoende afronding beskou.

Hierdie wantroue word nog verder in die geneesheer se toekomstige selfstandige ontwikkeling geprojekteer waar dit nou aanvaar is deur die Geneeskundige Raad, en daarom ook deur die mediese skole, dat buite die opleidingsentrum weinig kennis, vernuf, of verantwoordelikheid bekom kan word. Hierdie vlymende swaard swaai nou oor die hoofde van alle algemene praktisyns, ongeag die individu of die omstandighede.

Suid-Afrika se plattelandse struktuur sal nog baie jare lank stand hou. Die plattelandse geneesheer en hospitaal is 'n onmisbare deel van die mediese dienste in ons land, juis as gevolg van die besondere vaardighede wat in die plattelandse omstandighede tot ontwikkeling kom. Die Provinsiale owerhede se gesondheidsbeleid rus vandag baie swaar op die eregeneesheer poste wat deur algemene praktisyns in die platteland gevul word. Ek kan nie sien hoe hierdie kostelose dienste, en die manne wat dit lewer, onerkend kan bly of vervang kan word nie.

Ek huldig geensins die mening dat 'n algemene praktisyn alles kan doen wat 'n spesialis doen nie. Daar moet egter heftig beswaar gemaak word waar die spesialis die ware algemene praktisyn se terrein beset en eiendomsreg daarop eis. Soos sake nou staan, wek dit 'n droewige gevoel by die jonger praktisyn dat hy aan onwettige bedrywighede skuldig is wanneer hy sy aanleg en talente wil ontwikkel. By die ouer meer ervare man verwek dit slegs 'n ironiese laggie. Elkeen wat die mes opneem, het al, en sal weer daarmee struikel.

P. A. Rens

Hoofweg 335
Paarl
5 Oktober 1961

1. Briewerubriek (1961): S. Afr. T. Geneesk., **35**, 824.

FULL-TIME MEDICAL STAFF AND THE MEDICAL ASSOCIATION

To the Editor: Now that many of my colleagues have attended the biennial festivity of the Medical Association, which I unfortunately could not do this year, it is perhaps a good time to bring up the question of the relationship between the full-time medical staff employed by the Government, Provincial departments, the mines and municipalities — to mention most of them — and the Medical Association.

I am one of these full-time employees and I understand we total a third of all medical practitioners in South Africa. Without our contributions to the *Journal* and our support of Congress, I contend that both would collapse for lack of scientific material. Yet, if the Southern Transvaal Branch is any index of the position, very few of us are members of the Association, though we may find ways and means of attending Congress. The number resigning from the Association is increasing, and, in my opinion, the reason for this is basically that the Association has become mainly a trades union, interested in and caring for the pocket and the future of the private practitioner, and offering very little indeed to the full-time medical officer.*

If this is so, and I would welcome any convincing argument to the contrary, it is a sad state of affairs. The Association, if it is to continue to be a real force and a scientific and academic body of national importance, must actively seek to retain among its members the full-time professors, research workers, doctors and scientists who train our future doctors and lead the way in medical progress.

The full-timer asks himself why he should pay about R20 a year for the privilege of membership? As a doctor he would like to maintain his contacts with the body of the profession; on the other hand he feels that the Association does not help him or represent him, and its materialistic viewpoint is often directly opposed to his own.

Perhaps my full-time colleagues in the other Provinces are too lazy to worry about the position, and pay their subscriptions without a moment's hesitation, or possibly other branches of the Association are more attractive than my own, but I would put it to you, sir, that the problem is a pressing one. In Johannesburg there is a strong feeling that we should get away from the Association and found our own society. Do you want this to happen?

My own opinion is that full-time staff should be encouraged to remain. We should accept a large share of the responsibility for scientific meetings and organizations, but our monetary contribution should be nominal and our status unchallenged.

T. Randall

Baragwanath Hospital
Johannesburg
21 September 1961

[* The Medical Association is fully aware of the important problems which arise from the relationship of full-time medical staff to the Association. These problems and their implications were fully discussed at the recent meeting of the Federal Council of the Association which was held in Cape Town on 21 - 23 September. Federal Council is actively and urgently engaged in examining and re-assessing the whole position with a view to finding a formula which will make it possible for the Association to gain the wholehearted support and cooperation of every doctor in the country. See also the Editorial article which is published on p. 871 of this issue of the *Journal*—Editor.]

HEARTBURN IN PREGNANCY

To the Editor: In reply to Dr. Goddefroy's question[1] regarding the use of co-pyronil for heartburn in pregnancy,[2] I have no experience in its use.

The makers inform me that any activity that this preparation might have on the gastro-intestinal tract would be exerted by its clopane hydrochloride (cyclopentamine hydrochloride) content. It primarily produces peripheral vasoconstriction, as well as a slight vasoconstrictor effect upon the arterioles of the gastro-intestinal tract. In addition, it acts upon smooth muscle, producing relaxation of the intestinal masculature.

D. K. Quinlan

801 Provident Assurance House
Durban
5 October 1961

1. Correspondence (1961): S. Afr. Med. J., **35**, 744.
2. Quinlan, D. K. (1961): *Ibid.*, **35**, 628.

ISOLATION OF SCARLET FEVER PATIENTS

To the Editor: Having recently seen a few patients with scarlet fever, I am prompted to voice my opinion about our outmoded regulations with regard to the notification and treatment of infectious diseases. These regulations are governed by the Public Health Act No. 36 of 1919!

I feel that, especially with regard to the management of scarlet fever, these regulations are not only unsuitable, but even constitute a definite hardship to certain families. Scarlet fever as seen today is not the terrible illness it must have been in the days when these laws were promulgated. With adequate and early treatment the dreaded complications of rheumatic fever and nephritis are not often seen. Indeed, the incidence of these illnesses is definitely on the decline.

Let us examine what happens when a patient is diagnosed as having scarlet fever. One is legally obliged to notify this case to the local authority, i.e. the Municipal or Divisional

Council Health Department. Once the case has been notified, the patient *must* be admitted to the Isolation or Fever Hospital serving that area. Exceptions to this rule can be made at the behest of the local Medical Officer of Health, but in practice this rule is relaxed only where it can be shown that there are no other children in the house and that home conditions are suitable. The patient is then hospitalized for a minimum period of 4 weeks.

Are these stringent measures really necessary in the treatment of scarlet fever today? I maintain that they are not and would support that contention by drawing attention to 2 articles in the September 1961 issue of *The Practitioner*.[1,2] This issue is devoted to the infective diseases. Dr. Barber,[1] in a summing up of the infective diseases in general practice, stresses the fact that there has been a vast change in the severity of these diseases. He also points out the fact that it is only since the work of Griffiths and others (in the 1930s) in classifying the streptococci that we have come to appreciate that the rash, without which scarlet fever cannot be diagnosed, is far less important than the underlying streptococcal infection. He cites cases where, in accordance with public health legislation, the child with the rash is sent to hospital, while the brothers and sisters with nephritis and myocarditis have to be cared for in their homes. In an article on scarlet fever, Dr. Christie[2] states that there is 'rarely any *medical* need these days for sending the patient to hospital'. He states further that patients treated with penicillin become non-infectious within 24 hours. As far as exclusion from school or work is concerned, he maintains that, in view of the mildness of the disease as seen today, and because of the fact that streptococcal infections, equally infectious, but without the rash, do occur, such measures are only rarely necessary.

In view of these authoritative opinions, is it not time that our regulations were amended in some way? I am sure that many doctors are committing technical 'crimes' in not notifying scarlet fever. They do this because they feel it is not necessary to remove a child to hospital for 4 - 6 weeks for what is in most cases a mild illness, especially since such measures are not warranted in the light of present-day knowledge.

Norman Levy

210 Medical Centre
Cape Town
5 October 1961

1. Barber, G. (1961): Practitioner, **187**, 269.
2. Christie, A. B. (1961): *Ibid.*, **187**, 296.

POSTOPERATIVE SEPSIS

To the Editor: *Staphylococcus aureus haemolyticus* is responsible for much of the serious postoperative sepsis which has become so rampant. Suspicion is often cast upon the surgical team or the nursing staff of the ward.

Before making unwarrantable postulations and deductions, it is imperative that variables be minimized. In this respect, the patient himself might have to be indicted as the possible source of infection. Non-specific venereal disease is not a rare condition these days. Unlike gonorrhoea, it can be so mild as to escape the attention of the carrier — even in moderately severe cases, individuals are known to be unaware of anything untoward.

May it be intimated when a patient enters a medical institution, and especially the operating theatre, that every effort be made to exclude the above variable.

As an anaesthetist, I shudder to think that the anaesthetic equipment may also be a significant factor.

H. H. Samson

1110 Medical Centre
Johannesburg
4 October 1961

A NEW LOOK AT FIRST AID

To the Editor: It is with concern and regret that I have read your Editorial 'A new look at first aid', in the issue of the *Journal* for 30 September.[1]

The tenor of the article appears to imply that the present teaching by recognized authorities on first aid in South Africa is both out-moded and dangerous. In the case of the South African Red Cross some of the facts stated do not apply. It is interesting to note that the Editor considers that the methods employed in 'mine accidents' are good. The methods are those contained in the Red Cross First Aid Manual and all mine workers take the qualifying examinations of the South African Red Cross Society.

The control of haemorrhage sounds easy when 'a few turns of a crepe bandage applied over a pad can control any haemorrhage'. This, of course, is not always the case and Red Cross teaching is that there are occasions when this method will not control bleeding and then digital pressure or even a tourniquet may have to be applied. In the case of the tourniquet method, the manual emphasizes that it is dangerous and is only justified in the following circumstances:

(*a*) For severe haemorrhage threatening life.

(*b*) When digital pressure and the subsequent application of a firm pad and bandage have failed to control the haemorrhage.

(c) When the fatigue caused by long digital pressure leads to ineffective control.

It is maintained that the correct teaching must embrace all eventualities and the first-aider must know what to do if a simple method fails.

In the case of artificial respiration, the mental processes of the South African Red Cross Society have certainly not 'become ossified and immobilized by admiration for old-standing institutions', for it is the current teaching of this Society that mouth-to-nose or mouth-to-mouth where applicable is the method of choice. Full details and didactic films are in the process of being circulated to all Branches throughout the country. The Society has accepted the views expressed all over the world that as a first-aid measure, 'gadgets', such as tubes in expired-air-insufflation, may be dangerous except in skilled hands.

Confusion seems to exist in the mind of the Editor regarding real first-aid or emergency treatment and selected procedures by teams of operators in the field of fracture work. However admirable a Thomas splint may be, this can hardly be the first method advised for first-aid use by the masses. The first-aider cannot be taught that Thomas splints are necessary when he is called to a casualty with a fracture of a limb, in the country, in a street accident, or his home. It is essential to understand the principles of 'splinting' and how to achieve it by wooden planks or any other satisfactory improvized material. The textbook and teaching by Red Cross emphasizes improvization, but does not fail to describe the use of the excellent Thomas splint when this is feasible and available.

Little comment is required for the 'diffident' view expressed about teaching ambulance men to set up intravenous plasma drips — this is a specialized surgical technique and has no place in the accepted teaching of first aid in general.

I consider that a disservice will be done to the aim of teaching everyone first aid in South Africa if Editorials of this nature disparage the current methods in the way that has been done in the Editorial quoted above.

Lewis S. Robertson
Chairman

National Executive Committee
South African Red Cross Society
Johannesburg
5 October 1961

1. Editorial (1961): S. Afr. Med. J., **35**, 807.

[In writing the Editorial, our intention was certainly not to 'disparage the current methods' of first aid. In fact, we stated emphatically that 'the first-aid manuals, sponsored by such world-wide organizations as the Red Cross and the St. John Ambulance Association, have stood the test of time and have enabled these bodies to render splendid services'. The whole tenor of the article was to point out that, in spite of these 'splendid services', we should always be on the alert not to become smug and self-satisfied — especially in view of the impressive recent advances in our knowledge of traumatology. Our views were therefore intended to serve as the basis for an open and constructive discussion of our present position in the field of first aid, relative to the 'idiom' of the times in which we live—Editor.]